THE PRACTITIONER

JANUARY
1920

Grave Familial Jaundice of the Newly Born.

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BRIEF *Description.*—In grave familial jaundice, successive infants become jaundiced a few hours to a day after birth, pass into a drowsy condition, and usually die within a week, often with convulsions; it resembles physiological jaundice in its onset, and, like it, does not show any gross post-mortem appearances to account for the jaundice.

Pathogeny.—Pfannenstied regarded it as only an intensive form of the physiological jaundice, but not only is the prognosis very different, but there may be evidence of maternal toxæmia during the pregnancy. In three (Nason, Tylecote, Rolleston) out of the 25 family groups that I have collected—that is, in 15 out of 130 cases of newly-born infants with grave familial jaundice—the mothers had recurrent jaundice during their pregnancies. In the families reported by Smith and by Ritchie, the mothers had jaundice at the birth of one of the children, and in Arkwright's family of 14 cases with four recoveries the mother had had jaundice when four years old and was always "sallow." In Blomfield's cases, the mother suffered much from severe vomiting during the pregnancies, but this association appears to be exceptional. Against the view that this grave disease is due to maternal toxæmia, and that in its most complete form the mother is jaundiced as well as the child, it might be argued that, as a rule, the infants suffer much more than the mothers and that the jaundice does not appear until after birth; but it is obvious that similar objections might be raised to the transmission of congenital syphilis; and it may be added that in congenital obliteration of the bile ducts there is a similar or greater delay in the onset of jaundice. The following observation is perhaps in favour of the existence of maternal toxæmia :—

I gave the mother, who had had recurrent jaundice in three successive pregnancies with fatal familial jaundice in the infants, hexamine and salicylate of sodium in her fourth pregnancy; in the eighth month, she became somewhat jaundiced just before the birth of a boy who was never icteric and was well ten years later. During her fifth pregnancy she was treated by Dr. Spencer Lewis with small doses of hydrargyrum cum cretâ but was jaundiced during the eighth and ninth months of pregnancy, and bore a girl who was slightly jaundiced and died of acute summer gastro-enteritis when seven weeks old.

Benedict's cases of recurrent jaundice, with pruritus and hepatic enlargement, in two sisters during their pregnancies, these manifestations passing off after abortion or premature delivery and the offspring escaping icterus, might perhaps be regarded as incomplete cases and corresponding in this respect with the cases in which the offspring alone are jaundiced. It is highly desirable that the metabolism of the mothers of infants with familial jaundice should be thoroughly investigated, especially during their pregnancies. In the meanwhile, it is suggested that *in its most complete form the disease is hereditary, the mothers becoming jaundiced during the pregnancies of jaundiced infants, and that the usual familial form is an incomplete manifestation of the morbid processes.*

Ætiology.—Some of the families, in which the disease has been reported, are remarkable for the large number of pregnancies; in Arkwright's there were 15; in Auden's, 12; in Underwood's and Ritchie's, 11, and in Busfield's, Nason's, and Morris's, 10. Occasionally there are some miscarriages or premature births, but all the 11 pregnancies in the family reported by Ritchie went to full term. Some writers refer to the robust condition of the infants; but all Arkwright's cases were rather weakly, and one had an intra-uterine amputation of the forearm.

The disease appears to be less likely to attack the first and second born than the later infants in the families affected. The first-born escaped in the families reported by Arkwright, Smith, Auden, Busfield, McGibbon, Morris, Tylecote, Still (two families), and the first two children in those reported by Abt (two families), Duguid, Nason, and Ashby and Wright. The first-born therefore escaped in 14 out of the 25 families collected, and in several families the first-born was slightly jaundiced and was the only one to recover. There is no evidence that syphilis plays any part in the disease, and though infection has been put forward as a cause, there is no good reason to accept this.

The sex is not mentioned in some of the reports, but in those that I have collected there were 31 males and 31 females. The sex incidence thus differs from that of congenital hypertrophy of the pylorus and of congenital obliteration of the bile ducts, which are both, especially the former, much commoner in the male sex. It has occurred in twins (Still).

Morbid Anatomy.—There is bile-staining of most of the tissues, and, as in physiological jaundice, there is selective staining of the lenticular and other nuclei in the brain, while the cortex escapes. Punctate hæmorrhages in the viscera are described, but their importance is doubtful, for they are not uncommon in stillborn children. There is no gross obstruction of the bile-narrowing of the ducts which gives rise to obstructive cirrhosis. My own view is that a maternal toxæmia causes cirrhosis followed by a descending obliterative cholangitis; and Dr. S. Wyard suggests that there is an ascending cholangitis from the duodenum set up by the irritation exerted by the swallowed liquor amnii. Most writers agree that all cases are not necessarily due to the same mechanism.

The Severe Infective Forms of Jaundice, such as those due to umbilical, cutaneous, or intestinal infection, formerly occurred in epidemics, and were responsible in the old insanitary lying-in hospitals for a terrible mortality, but, as far as I know, are rare now. The jaundice comes on about the fifth day of life, or later than in familial jaundice, and is accompanied by signs of grave septicæmia, high fever, and hæmorrhages especially from the umbilicus or the alimentary canal. Winckel's disease (epidemic hæmoglobinuria with bronzing of the skin) and Buhl's disease (acute fatty degeneration) are manifestations of this septicæmia of the newly-born, the infection probably entering from the intestines. The prognosis is very grave. Treatment should consist in the administration of antistreptococcic serum, washing out the bowel, and the oral administration of intestinal antiseptics such as minute doses of calomel.

In Melæna Neonatorum jaundice is often present; thus Pitfield records as hæmophilia four successive males with jaundice and hæmorrhages.

Congenital Splenomegalic Hæmolytic Jaundice runs a mild course, and can hardly be called a disease; it is recognized by the characteristic fragility of the red blood corpuscles when tested with hypotonic solutions of salt. *Congenital syphilis* does not often cause jaundice; it usually comes on early and is accompanied by other signs of the disease such as enlargement of the liver and spleen. Secondary pyogenic infection may occur in these cases.

Treatment.—On the hypothesis that grave familial jaundice of the newly-born is due to fœtal toxæmia of maternal origin, the prophylaxis should consist in care of the mother's diet and the administration of intestinal and biliary antiseptics, such as hexamine, salicylate of sodium, minute doses ($\frac{1}{20}$ gr. of calomel), tetrachloride of naphthaline, salol, and guaiacol. Ballantyne recommends chloride of calcium.

The infant should be treated with small doses of calomel, which some authors consider to have done good, and should not be given the breast.

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remainder of his evidence will, in consequence, have an enhanced value.

Nothing pleases cross-examining counsel better than to make a witness angry. Cross-examination is intended to demonstrate your want of candour or your lack of knowledge. Do not be offended if counsel insinuates that you are a gross perjurer, or if the judge in summing up infers that you are either very unobservant or are much misguided. Remember that the case must go against someone, and that if you have these pleasantries levelled at your head, your medical friend who is opposed to you has been saved what he possibly might have been foolish enough to take seriously.

The medical witness should take pains to articulate distinctly and slowly; he should look at counsel when he is asked questions, but address his answer to the judge and jury. He should take care not to outstrip the judge if he is taking notes. Watch the judge's pen. The exact question put should be answered before attempting to mention other details or explanations which are considered necessary. What is put before the Court should be stated in simple, non-technical language.

Under the Workmen's Compensation Act, a workman *must* submit to a medical examination "provided and paid for by the employer." If he refuses, his compensation under the Act may be suspended. There is no such obligation in actions for damages at Common Law or under the Employer's Liability Acts, but there is seldom any difficulty in arranging a medical examination, for a refusal would seriously prejudice the plaintiff when the case came for trial.

In all such examinations it is wise—apart from obvious etiquette—to arrange for the plaintiff's doctor to be present; but remember the examination is not a consultation. As the plaintiff's doctor will probably be in communication with his patient's lawyers, a sphinx-like attitude of detachment at the examination and a judicious concealment of your views from all concerned is often your duty.

One of the most frequent mistakes made by doctors unaccustomed to these examinations is to put what lawyers call "leading" questions. If, for instance, when the question is whether the internal semilunar cartilage of a knee joint has been dislocated, the plaintiff is asked whether the joint "locked" at the time of, or soon after, the injury, a suggestion is made which may be improperly used.

Some time ago I was asked to examine and report upon a man who alleged epileptic seizures as the result of an accident. He was an impostor, but gave a clear, connected account of his seizures. He had been examined by many doctors on behalf of an insurance company, and a perusal of their reports showed that he had gathered the physical signs and symptoms of epilepsy from the leading questions put to him by one doctor after another. The first doctor asked if he frothed at the mouth; he told the second examiner that he did, who in turn asked if he had bitten his tongue. The third doctor was informed that he frothed at the mouth, that he bit his tongue, and that it had always bled profusely, and so forth. At my examination he went still further, alleging that he had bitten his tongue so badly that he had to live on slop food for ten days. I was able to prove this to be untrue by closely questioning his wife as to the food she had cooked for him.

If previous medical reports have been made, they should be carefully perused, and, if not supplied, should be asked for and obtained before the examination is made. It is, of course, advisable to retain an exact copy of all reports you make.

Insurance companies are not accustomed to technical medical terms, and doctors should remember that all reports should be written in plain, businesslike language. Insurance companies know how to deal with gentlemen who, as a young consultant recently did, attempt to create an impression by saying "Nephrolithiasis has induced a condition of Pyonephrosis," when all that is meant is that the kidney was being worried by a stone in its inside. I have before me a long report from a young consultant, four pages of which detail the result of a physical examination made by him, in which he states that diplopia and nystagmus were absent, that Romberg's sign was negative, that the plantar response was definitely flexor, that the rotatory test for cerebellar trouble gave negative results, and that there was no dysdiadokokinesia. In forwarding his report to the Insurance company, I indicated that all that the first four pages of close type amounted to was, that there were no physical signs of disease. This, I take it, would be much more to the liking of busy business men who are neither interested in nor awed by technicalities.

The only way of getting at the history of an accident is to ask the injured man to tell his own story. A brief description is all that one can listen to, and he must, therefore, be guided out of irrelevancies. At best, the working man's vocabulary is very limited, and he invariably credits everyone with a knowledge of the technicalities of his particular craft, which few possess.

Sometimes an otherwise cunning rogue gives himself away by twisting and turning himself into grotesque attitudes, in an endeavour to show how the alleged accident occurred. This is often useful, for his free movements are alone presumptive evidence of a return to health.

Lawyers say that circumstantial evidence is often more valuable than direct, and so I believe. The method adopted by a malingerer or a neurasthenic in narrating his symptoms, is often more informing than abnormalities found on physical examination.

A great deal may be learned by watching patients dressing and undressing. Some are ceremonious with regard to details, and often ostentatiously almost demand assistance in the removal of their clothes. It is best to humour them in the process of undressing. They generally dress themselves after the examination.

Morbid introspection is easily implanted on one who, accustomed to do laborious work, suddenly finds himself unoccupied and living in comparative ease on club money, whose environment is suddenly converted into a sympathetic one, and whose new circumstances in almost every detail present a vast contrast to his ordinary every-day life before the accident. What wonder if he misinterprets various bodily stimuli, which would in his ordinary daily life have been ignored?

It is, nevertheless, our duty, not always an easy one, to preserve an unbiassed attitude and to maintain throughout a judicial impartiality. Many litigants, indeed most, come to an employer's medical examiner with a preconceived idea that he is their enemy.

(*To be continued.*)

Venereal Diseases as we see them To-day.

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GENERAL.

SIX years have now elapsed since the memorable meeting in the Albert Hall, and it would be well, in my opinion, to sum up the successes which have been gained and the failures experienced during this period. It is only by recognizing and correcting these failures, that we can ultimately reach the goal at which we have aimed.

The propaganda work has succeeded in opening the eyes of the public to the dangers of venereal diseases, with the result that patients come up for treatment at an earlier stage than formerly. It has failed to reduce the incidence of venereal diseases. The vivid and exaggerated pictures which have been drawn of the rarer sequelæ of these diseases have given the public false impressions, and victims have become so terrified that there has been produced a venereal neurasthenia, which is infinitely more difficult to cure than the actual disease itself.

Prophylaxis has engaged the attention of many, and is now relegated to a position subordinate to moral training. Although the prophylactic measures used have saved many from infection, at the best it was only shutting the stable door after the horse had been stolen. It was a pity to issue "prophylactic packets," when micturition and washing with soap and water immediately after connection are simpler and just as efficacious. Possibly because of the simplicity of this teaching, which hailed from the medical school of Salerno four or five centuries ago, it was thought that more reliance would be placed by the public upon special outfits. Because mercury has been used for syphilis since the disease came to Europe, when it was confounded with scabies, which had been treated with mercury for hundreds of years before, and because Metchnikoff had found that rubbing calomel ointment into a scarified wound on a monkey's forehead—an insult to which the penis is seldom subjected—prevented infection with syphilis, and because potassium permanganate has been used in the treatment of gonorrhœa for so long, it was thought that the outfits should contain both calomel and potassium permanganate. As a matter of fact, tubes containing a semi-liquid colloidal mercury cream, and supplied under the name of *serenol*, are equally as good and certainly more convenient than the dual outfits. In the first place, it is one thing to prevent organisms from growing but quite another to kill them when grown. In the second place, it was never advised that surgeons should sterilize their hands with that particular antiseptic which (in a test tube) most easily killed the organism they were going to meet. In the third place, surgeons have recognized for years that in sterilizing their hands before performing an operation, washing with soap and water is the most important step. In other words, with our prophylactic packets we have traversed the same ground which we trod between the antiseptic period of Lister and the aseptic period of to-day, without noticing the reiteration.

Moral teaching may be all very well in theory, but it will not be very productive of success in practice, unless propaganda becomes extended so far as to reach every individual, not on one occasion only but on several.

Let us now turn our attention to another aspect of the problem which has emanated from the anti-venereal legislation, namely, free treatment. This, in the first place, means that each taxpayer is paying for another's misdemeanours and, may be, the innocent for the guilty. So far as the Lock Hospital is concerned, I never thought free treatment would be a success—an opinion which I hold now more strongly than ever. We have not the same control over the patients as when they paid, be it ever so little, and patients are not so regular in their attendances as they used to be. The more uneducated the man is, the less value he places on anything he gets for nothing; while the educated take advantage thereof, and come for a treatment which they could well afford to have in private.

The disadvantages following free treatment are by no means only on the patients' side, for the institutions, to get as big grants as possible, are not guiltless of *finesse*. For instance, if a patient is examined, that is one visit; if he has an instrument passed *intra urethram*, it is another visit, and the same if he has an injection or a pathological examination. In fact, the same patient can be reckoned as four visits each time he appears, and so it becomes disadvantageous to cure a patient too quickly.

As pathological examinations are well paid for, an enormous number of unnecessary ones is made. Also, pathology being the path of least resistance, the medical officer, who is often not too well versed in clinical matters, resorts to such examinations to the detriment of clinical medicine. From this follows the still further disadvantage that a patient believes in nothing but the result of a pathological test. Another great disadvantage resulting from the free treatment is, that most of the institutions have come within the jurisdiction of a department which is attempting to standardize treatment and pathological tests, with consequences which must be disastrous to all concerned. The moment standardization becomes paramount, healthy competition is removed and a stationary state produced instead of an advancing one, which is contrary to the fundamental law of the universe. Medicine of all sciences is the least capable of being standardized, because at present it is the least exact science and certainly the most illogical.

This brings us to the point whence we can view the pathological domain of venereal diseases, upon the foundation of which the whole of the organization of the venereal problem has been raised. It is the realization of the instability of this structure which will cause the public to be our judges.

An illustration or two of this argument will make it clearer. To-day patients demand a blood-test after their course of arseno-benzene, and, if the result is negative, refuse to take the intermittent treatment, which is the most important part of the programme. Most patients demand a blood-test every six months, and prefer at first to interpret the results themselves; but when later they get two negatives and two positives or develop a clinical recurrence with a negative test, you are asked askance what it all means. Many patients suffering from gonorrhœa demand a smear before undergoing a urethral and rectal examination, and are prepared at first to pin their faith upon the result. Men who have had syphilis, and who, on clinical grounds, have been legitimately allowed to marry, have committed suicide on finding after the event that their blood was positive. I do not hesitate to say that venereal pathology, as at present constituted, is a curse to both patient and doctor.

No mention is made, throughout these six years, of the clinical diagnosis and treatment of venereal disease, a knowledge of which will do more to save the suffering of the infected than all the pathological tests put together, however perfect they might become, not to mention the pocket-saving to the taxpayer. Having combined my clinical with my pathological work, being one of the first to do the tests now in use, and having spent hours a day for several years with the hope of being able to unravel the mysteries of such tests, I will tabulate here the results of the former and will refer in more detail below to the results of the latter.

1. The *Spirochæta pallida* can be demonstrated by most observers in only 60 per cent. of chancres.

2. A negative Wassermann reaction means nothing, while a positive is only strong presumptive evidence that the patient has had syphilis. A positive reaction does not necessarily mean that the lesion in question is syphilitic, nor that the patient is actively syphilitic, nor that he requires treatment. A positive reaction may be an indication of the patient's protective action against the disease.

3. Owing to the varied morphology of Ducrey's bacillus and to the difficulty encountered in demonstrating it, scraping a soft sore is in most cases futile.

4. In sub-acute, chronic, and recurrent cases of gonorrhœa, in both men and women, the gonococcus cannot be demonstrated in more than 15 per cent. of the cases.

5. A positive complement fixation test in gonorrhœa is frequently an indicator of the patient's resistance against the disease, but it by no means signifies that the disease is active or that the patient requires treatment.

A clinical diagnosis of any visible lesion should not be accompanied by so much as a 1 per cent. error.

Clinical observation and experience are the only reliable guides when it comes to a question of deciding whether the patient has an active lesion or not; whether further treatment is required or not, and whether a patient is fit to marry or not. As a very large number of syphilitics, for the remainder of their earthly existence, give a positive Wassermann reaction, which is uninfluenced, or only temporarily influenced, by treatment, more good can be done by telling the patient that the reaction is useless as a guide to treatment than by prescribing an unlimited number of courses of arseno-benzene. To-day, venereal diseases are suffering from being over-treated; especially is this the case with gonorrhœa. Over-treatment for syphilis has two dire results, (1) metallic intoxication; (2) nervous implication. These two complications have become of such daily occurrence, the clinical pictures presented are so diverse, and mistaken diagnoses are so frequently made, that I must refer the reader to a full account of them.[1]

METALLIC INTOXICATION.

All metals when they act as poisons produce the same symptoms, although one condition may be more readily brought about by one metal than by another. Mercury most readily causes stomatitis, but arsenic frequently does so, a fact not generally known. Both mercury and arsenic produce intestinal congestion, polyneuritis, hepatitis, and nephritis, although we are more accustomed to meet with jaundice, acute yellow atrophy, and uræmia caused by arsenic than by mercury. Mercury seldom produces a dermatitis, while arsenic frequently does so. Both mercury and arsenic can produce endothelial degeneration, as witnessed by hemiplegia and paraplegia setting in suddenly after a course of arseno-benzene.

When metals act as slow poisons, the changes which take place in the organs are mesenchymatous rather than parenchymatous, *i.e.*, the supporting tissue in contradistinction to the cellular is attacked. Mesenchymatous changes are unfortunately degenerative and progressive before they are recognized. The mesenchymatous changes produced by metals do not differ from those wrought by any chronic poison or from those which naturally occur *in corpore* when a disease becomes chronic. Every reader is aware of the empirical use of iodine when there is fibrous tissue formation, *i.e.*, in the chronic stage of most diseases. As I have shown that iodine acts by increasing the sulphur metabolism, and that the latter is the body's sheet anchor when the above-described condition of affairs exists, it now becomes clear why I have continuously advocated colloidal iodine and intramine in the chronic stage of most infections, in preventing metallic poisoning, and in overcoming it if in being. Since I advocated intramine in arsenical poisoning, others have employed ordinary sulphur, colloidal sulphur, and ichthyol, which are valueless.

Why is it that no sulphur preparation so far prepared is as active as intramine? The activity of metals and non-metals depends not upon their chemical but upon their physical action. For a metal or non-metal to exert its full action it is necessary for it to be in such a condition as to be readily taken up (adsorbed) by the protein particles (resisting substance or antibody), the action of which it is going to accelerate. There is no such thing as killing organisms *in corpore*, directly, and, in my opinion, phagocytosis of living bacteria never occurs in the body but only in the laboratory.

Protein, owing to its unique chemico-physical molecular configuration, will most readily take up another body similarly constituted, and then bodies which have a large molecule containing amino-groups (NH_2), especially if such amino-groups are in the ortho-position to the active element. In other words, when intramine is used much more sulphur is taken up by the protein particles than from any other preparation of sulphur. This is the reason why arseno-benzene is so much more active than Fowler's solution, colloidal arsenic, or atoxyl. There is greater need to employ the best remedy at the earliest possible opportunity, when the body is suffering from a chemical poison, than when it is suffering from a disease. Therefore, to prescribe ordinary sulphur in the place of intramine, in the case of metallic poisoning, is like placing a gun without cartridges in a man's hands and then expecting him to bring down birds.

There is scarcely a biological or industrial so-called chemical reaction in which the physical action is not more important than the chemical. In the former, we have the excellent examples of intramine—a non-metal—overcoming metallic poisoning, and colloidal manganese—a metal—overcoming mustard-gas poisoning. It was the curative effect which metals exerted in yperitis, which forged the final link in my theory of oxidation and reduction. In industrial chemistry, we have the action of sulphur—a non-metal—in the vulcanization of rubber, and the action of manganese—a metal—in the drying of oils. In addition, mention may be made of the action of metals in the polymerization of butadiene to synthetic rubber, the hardening of liquid fats and fatty acids by hydrogenation, the production of light by means of incandescent mantles, etc.

In all these reactions the element acts catalytically, *i.e.*, as a substance which changes the velocity of a reaction without itself being changed by the process. Broadly speaking, catalysts are colloidal, which is only to be expected, for a colloid is a dynamical state of matter in contradistinction to a crystalloid which is a statical condition of matter. Medical men of the present day, owing to the bustle of life which gives them little time for reading, are apt to regard a subject as beyond their reach if the word colloid appears therein. This is a misfortune, because, as every drug has to become converted into the colloidal state before it can exercise its action, no important advance will be made in the future unless the purely chemical action is made subordinate to the colloidal and physical actions. The whole subject of chemotherapy, the whole subject of immunity, the whole subject of staining, etc., are in the main physical actions exhibited by colloids, actions which are easy to comprehend and actions which reduce the three from mystery to simple fact.

Just as mercury came to be regarded as a specific for syphilis because syphilis was thought to be a modified form of scabies, so did arsenic come to be used because the causative organism of syphilis was considered to belong to the same group of organisms as the trypanosome of sleeping sickness. Neither mercury nor arsenic are specifics for syphilis: first, because there is no such thing as a specific; secondly, because mercury acts in virtue of its atomic weight, producing an effect inferior to that obtainable with thorium (Hg. 200·6 Th. 232·4); thirdly, because metals act as oxidizing agents of which mercury and arsenic are not good examples; and fourthly, because their action is only so pronounced because there are more protein particles in circulation to which they can become adsorbed in syphilis than in any other disease. Another reason why arsenic was used was because it is one of the easiest elements to work with, for it can act both as a metal and as a non-metal. Furthermore, the exclusive use of metals completes only half of the chemotherapeutic programme, because, based as it is on oxidation and reduction, it is impossible to obtain the best results unless the dual action is brought into play. As the reducing action of the protein particles is regulated by non-metals and in all living cells by sulphur, the reader can now appreciate the boon the introduction of intramine has been.

As sulphur is the non-metal acting *in corpore*, intramine becomes *ipso facto* non-toxic. As iron is the metal regulating the oxidizing action *in corpore*, and as manganese is more closely related thereto than any other metal, it is now clear why I should have advocated the use of these two metals, which are non-toxic compared with such metals as arsenic, antimony, and silver. Because intramine will prevent and overcome the toxic action of metals, it has been supposed that its use with arseno-benzene and mercury would lessen the action of the metal. This is not so. It is true that, in industrial chemico-physical reactions, the presence of a non-metal may interfere with the catalytic action of a metal and *vice versâ*. But here one is dealing with a single action. In the body, owing to the fact that protein has both a positive and a negative electric charge, an action exhibited thereby can be of a dual nature. As an element does not act as a poison until its maximum therapeutic effort has been spent, and as a parallel exists between oxidation and reduction and the dual action of protein, the simultaneous use of metals and non-metals is not contra-indicated, but, on the contrary, strongly indicated. Intramine will not only increase the therapeutic action of arseno-benzene and mercury, but it will also prevent metallic intoxication and cause the symptoms of such to vanish if it is prescribed quickly enough. Finally, its action is increased by iodine.

Since I have included intramine in the treatment of every case of syphilis, I have not had a single instance of metallic poisoning, within a period of nearly five years.* In all early cases of syphilis, 2·5 ccm. of intramine should be injected intramuscularly after the fourth or fifth intravenous injection of arseno-benzene, either once or twice. In all late cases of syphilis, one or two injections of intramine should precede any treatment with metals. If the case of metallic poisoning is urgent, 100·0 to 300·0 ccm. of colloidal iodine should be injected intravenously, and at the same time 2·5 ccm. of intramine should be injected intramuscularly into each buttock. The three injections are repeated in five days, if necessary. If the case is not urgent, colloidal iodine can be prescribed internally (ʒ iij *ter in die*) and 2·5 ccm. of intramine injected intramuscularly every five days until the trouble has vanished.

NERVOUS SYPHILIS.

For the past eight years I have attempted to draw the attention of the profession to the increase of nervous syphilis, to the factors responsible for it, and to the way in which it can be checked. If the increase continues in the ratio it is now doing, syphilis will soon have to be regarded as a nervous disease. It may be taken as tolerably certain that in every case in which the organisms become generalized, they do so almost as thoroughly and at about the same time in the nervous as in the systemic part of the body. Although their generalization in the nervous part may produce neither signs nor

* Since the above was written, I have just had a case which presented a mild toxic jaundice, which quickly vanished under another injection of intramine

symptoms, any late manifestation is not due to a fresh entrance or generalization, but is dependent upon the first which, in my experience, approaches its height about the eighteenth week after infection.

As drugs do not so readily pervade the nervous as the systemic part of the body, and as the protective substances (antibody) circulating in the blood form a very strong pillar of support for the nervous part, it can be readily understood that this pillar must *ipso facto* cease to exist as soon as the protective substances are destroyed in the systemic part. As the action of treatment is to destroy the protective substances, and as the systemic part is sterilized before the nervous part can be, it stands to reason that a stimulus is given to the organisms in the nervous part to multiply to their heart's content. This is the reason why we see early nervous syphilis most frequently between the eighth and sixteenth week after the completion of a course of arsenobenzene and mercury. Note that this is the same time as recurrent chancres appear, so often diagnosed wrongly as fresh infections.

The organisms apparently reach the nervous system *viâ* the blood vessels, when they involve the meninges (mainly dura) and nerve tissue proper, and *viâ* the choroid plexuses and meninges by filtration, whereby they circulate in the cerebro-spinal fluid, and involve the meninges (arachnoid and pia) and the nerve tissue proper. The various routes along which the organisms travel and the various structures involved explain the protean character of nervous syphilis. As, broadly speaking, no two cases of nervous syphilis are exactly alike, it is difficult to imagine that there is a special strain of organism which shows a particular affinity for nerve tissue, a belief which becomes still less probable when nearly every nervous lesion has or has had a vascular or meningeal origin. Vascular lesions in the brain lead in early syphilis to hemiplegia and *Encephalitis hæmorrhagica*, and in late syphilis either to encephalomalacia, if the degeneration is primarily vascular, or to degenerative encephalitis (G.P.I.), if the degeneration is primarily nervous, the latter resulting when the organisms develop extramurally. In the cord we have, in early syphilis, paraplegia, and in late syphilis either myelomalacia, evidenced by chronic anterior poliomyelitis and amyotrophic lateral sclerosis, or degenerative myelitis (tabes).

Meningeal lesions in early syphilis remain either wholly meningeal, or cause, by adhesion to and compression on the subadjacent nerve tissue, meningo-encephalitis, of which the most common symptoms are epilepsy and cranial nerve palsy and meningo-myelitis, of which the best clinical example is an irregular Brown-Séquard syndrome. In late syphilis, meningeal lesions (arachnoid and pia) cause degenerative encephalitis (G.P.I.) and degenerative myelitis (tabes). In early syphilis generalization is the rule, and in late syphilis, localization. In early syphilis, an involvement of the arachnoid and pia leads to a generalized leptomeningitis. In late syphilis, it is natural to suppose that the organisms would settle down in certain favoured spots, such as in the termini and deepest parts of the sub-arachnoid lake. Clinically, they seem to do so, as evidenced by (1) optic atrophy—terminus of a sub-arachnoid space, (2) third nerve palsy—deep interpeduncular space, (3) cervical tabes—terminus caused by bulging produced by cervical plexus, (4) lumbar tabes—terminus caused by bulging produced by lumbar plexus. In most cases of degenerative myelitis (tabes) the primary lesion is in the posterior nerve roots—termini of the sub-arachnoid space. All these lesions are most likely to occur if the systemic part of the body is sterilized by treatment and the nervous part is not. If such a state of affairs is produced in early syphilis, the lesions will be mainly meningeal and at first non-degenerative, while, in late syphilis, they will be mainly nervous and degenerative.

The neuro-recurrences occurring in early syphilis recover under further and appropriate treatment, but a certain percentage develop a degenerative lesion later, and they are most apt to occur when no intermittent treatment for one or two years follows the initial treatment with arseno-benzene and mercury. The late neuro-recurrences are met with when a man in a perfect state of health and in the dormant stage of the disease is made to undergo a course of treatment merely because his blood is positive.

How can this increase of nervous syphilis be diminished? It can be diminished by remembering three rules: (1) The intermittent treatment for two years is more important than one or more courses of arseno-benzene and mercury; (2) the use of metals only, by depressing oxidation and thereby depriving the nerve tissue of the oxygen it most urgently requires, renders the nervous part a *locus minoris resistentiæ;* (3) never treat a man in the latent stage, if he is clinically sound, if his blood or even his cerebro-spinal fluid is positive. Since I have incorporated intramine in my maximum course of arseno-benzene, and have proscribed intermittent treatment by mercury, intramine, and iodine afterwards, I have, in the course of four years, only had one neuro-recurrence—a case of unilateral optic neuritis. Within the same period in cases not so treated, I have come across over 100 cases of neuro-recurrence. Being frequently asked if the present-day tratment has diminished the incidence of tabes and G.P.I., I should like to state that, in my experience, both are more common and set in earlier than used to be the case. Caution should be observed in advising a man to undergo treatment merely because his cerebro-spinal fluid is pathological, because (1) the fluid may become normal without treatment and without nervous symptoms arising; (2) treatment may render such a fluid nearly normal, but, in so doing, it may precipitate an incurable lesion.

SHOCK.

Shock[2] is a subject which must interest all who employ intravenous medication, besides being one which has been rendered well-nigh incomprehensible by the words, etc., which have been coined to explain its *rationale.* The clinical picture is too well known to need description here, so it will be best to open the subject by stating that shock, anaphylaxis, allergy, hypersensitiveness, nitritoid crisis, etc., are all words for the same phenomenon—a phenomenon which results when certain changes take place in the colloidal character of the protein particles. These changes are, as a rule, wrought by an increase of "acidness" or "alkaliness," to use two simple but horrid words, of the blood or by a direct tacking on (adsorption) of another colloid (protein) perfectly emulsoid. The changes which take place in the protein particles are (1) precipitation with increase in the size of the particles; (2) going into true solution. Both diminish and may even prevent the normal functions of the particles, the chief of which are those of oxidation and reduction.

If a drug like aluminium hydroxide is injected intravenously into a rabbit, death takes place instantaneously; so, too, if silicic acid is injected. If both are mixed before and then injected, or if both are injected simultaneously, one into one ear and the other into the other ear, death does not occur. When death occurs, complement vanishes from the blood. The aluminium hydroxide increases the "acidness" of the blood (raises the hydrogen ion concentration), while the silicic acid does the opposite. As death does not occur when the substances injected are neutral, and complement does not disappear when this state is reached, it is perfectly clear that complement is the balance between the "acidness" and the "alkaliness," *i.e.*, is the normal hydrogen ion concentration or standard alkalinity of the serum. This equilibrium has to be preserved if the protein particles are to fulfil their normal functions. Death is prevented following aluminium hydroxide, if intramine is allowed to precede or quickly follow it, while in the case of silicic acid death is hastened. Intramine being non-metallic increases the "alkaliness" and therefore neutralizes the "acidness" caused by a metal and only aggravates the condition caused by another non-metal, silicon. Therefore a patient shocked by arsenic or mercury will, and does, as I have been able to prove, instantaneously recover if an intravenous injection of intramine be prescribed quickly enough.

WASSERMANN REACTION.

When I found that aluminium hydroxide alone or silicic acid alone, but not both when mixed, would in the presence of complement alter the surface and thereby the permeability of the protein particles, and having learnt from my previous experiments that this is what takes place in a hæmolytic system when amboceptor and red blood corpuscles meet in the presence of complement, resulting in hæmolysis, it occurred to me that, if my view was correct, either colloid alone should be able to replace the amboceptor. I found that such was the case, which proved at once that what occurs in the test tube is the same as what takes place in the body in the condition of shock. As hæmolysis is only one way of exhibiting the same colloidal phenomenon witnessed in all immunity reactions—agglutination, precipitation, etc. —it shows that immunity, anaphylaxis, chemotherapy are all different modes of exhibiting the same changes which take place when one colloid like protein meets another. If this is so, why does a syphilitic and not a normal serum give a positive Wassermann reaction, and why do chemo-therapeutic drugs cause symptoms to disappear more quickly in syphilis than in any other disease? The answer to both is the same; it is simply a question of degree or intensity of the exhibition of the phenomenon above referred to. In a syphilitic serum, there are more protein particles than in any other diseased serum; consequently, the physical action revealed by it will be increased in intensity, just as it would be more striking to see in an enclosed space a hundred men rather than one succumb to the dose of gas necessary to kill the former. In the Wassermann reaction, the increase in the number of particles in a syphilitic serum, which undergo a physical change during the time elapsing between drawing the blood and performing the test, is sufficient to upset that neutrality (complement) which is necessary for hæmolysis to occur, consequently, the red blood corpuscles fall to the bottom of the tube, leaving the liquid above clear (positive reaction). In chemotherapy, the increase in the number of particles enables more of the metal prescribed to become attached to the protein particles, the action of which it stimulates and accelerates.

Unfortunately, in practice everything is not so simple, for the protein particles when they leave the body can undergo certain physical changes over which one has no control and cannot detect. As these changes may materially modify the result of the complement fixation test, it follows that a negative reaction is always valueless and a positive one means no more than that presumably the patient has had syphilis. No one has a right to draw any other inference. It may be of interest to point out that the results obtainable in the Wassermann reaction, and this applies to all immunity reactions, are cyclical, *i.e.*, there may be just as close a relationship between the most positive reaction possible and a negative reaction as between a negative reaction and one only slightly positive. One of the fundamental laws governing the interaction of colloids is, that an excess of either tends to nullify the usual effect produced when the maximum quantity necessary to bring this about is not over-reached. As good illustrations of this law, mention may be made of the fact that a negative Widal is the rule in the worst cases of typhoid (agglutination); that a negative Wassermann reaction may be obtained in the worst cases of syphilis, because the stage following the ultra-positive reaction is a negative reaction (complement fixation test); that large doses of an ordinary shock-producing substance will not affect the animal (anaphylaxis), and that large doses of chemo-therapeutic agents and vaccines are less likely to be followed by untoward results than when small doses are used (chemo-therapy). The truth of the last statement can be practically demonstrated in the use of arseno-benzene. Gradually increasing doses are more likely to be followed by certain toxic manifestations than when maximum doses are employed from the start.

VACCINE-THERAPY.

To get the best results with either chemo-therapy or vaccine-therapy, it is important to have continually in mind that chemo-therapy influences the surface of the protein particles, while vaccine-therapy influences their bodies or substrata. Chemo-therapy aims at increasing the oxidizing action of the protein particles, the actual factor which brings about the parasite's death. As it is extremely easy to overstep the action desired in both chemo-therapy and vaccine-therapy by injudicious use of the various agents, and as the intensity of the surface action will naturally depend upon the state of the substrata of the protein particles, the reader can see that there is an intimate relationship between the two. Practice shows that it is easier to overstep the mark with metals than with non-metals, and in diseases which are localized than in those which are generalized. Therefore, in such a disease as gonorrhœa, so long as it remains localized to the urethra, satisfactory results can be obtained only when each case is treated individually, and when the greatest attention is paid to the dose and time of its repetition.

In vaccine-therapy, owing to the fact that an immunity is rapidly produced which, if over-reached, will nullify any beneficial result obtainable (excess of colloid), a disappointment which all have suffered who have to do with the immunizing of rabbits against the red blood corpuscles of a sheep for the amboceptor in the Wassermann reaction, when just one more injection may reduce a perfect immune serum to one which has no immunity. Moreover, a general focal and local reaction (mild shock) is apt to occur with any vaccine however non-toxic it may be said to have been rendered, and this, if it does occur, reduces the degree of immunity capable of being produced. Both these disappointments are most apt to arise if too large doses are given and if too long intervals are allowed to elapse between the injections.

As an overdose of a metal may lead to its own oxidation, whereby an increase in the "acidness" of the blood is brought about, it is easy to see that the discordant results obtained in these two important therapeutic branches are due to the fact that the prescribers are not *au fait* with their *modus operandi.* It must be remembered that both chemotherapy and vaccine-therapy aim at increasing the action of the patient's protective substance (protein particles). As a patient who is very run down may have little or no protective substance, the use of either drugs or vaccines may have the same result as goading a horse to further effort when it has already expended its maximum. This is the reason why mercury and arseno-benzene may do more harm than good in cases of malignant syphilis, and why in gonorrhœa the untimely use of a drug or vaccine may precipitate a complication, or may even make the disease become generalized. As the complement fixation test in gonorrhœa has been revived, and as it was thought anew that it might be employed as a test of cure and as a regulator of treatment, it will be advisable to deal with this part before proceeding with the practical application of the subject in hand. Working with Klein[3] on this very subject eight years ago, and having applied the test since to several of the cases then treated with sensitized vaccines intravenously, I have come to the conclusion that the test is useless in both respects, for the following reasons :—

1. A positive test obtained after any vaccine is a sign of the immunity produced against that vaccine, but not necessarily against the disease—*vide* the onset of arthritis after vaccine-therapy, when the maximum degree of positivity or immunity has been produced.

2. The reaction past the most positive obtainable is negative.

3. Clinical relapses are most common when the serological reaction is negative, and recurrence of a positive reaction is not usually accompanied by a clinical relapse. Therefore a positive reaction is often a contra-indication for treatment, and a sign of the

patient's good protection against the disease.

4. A positive reaction produced by vaccines not only fails to indicate the degree of immunity against the disease, but it also fails to indicate whether the vaccine should be repeated or when it should be repeated, because the time varies in different individuals from the rise of a beginning positive reaction to the summit of a completely positive reaction to the fall of a negative reaction.

We must now compare sensitized vaccines with Thomson's detoxicated vaccines and both with pallamine, trimine, and intramine. Sensitized vaccines in plain urethritis were not of much value, but in the complications of gonorrhœa, when used fresh and intravenously, they were better than any formerly or since produced. Sensitized vaccines are troublesome to prepare, and they quickly deteriorate on keeping, consequently Thomson's vaccine, which is different in these respects and far more therapeutically active in plain urethritis, has marked an extremely important advance in vaccine-therapy. Therefore, so far as vaccines go, Thomson's detoxicated gonococcal vaccine is the best obtainable. It is difficult to compare vaccine-therapy with chemo-therapy, while the one is an adjunct to the other, but, broadly speaking, in my hands, using chemo-therapy alone and vaccine-therapy alone, better results have been obtained with the former than with the latter in cases of urethritis (first attack), in the acute stage of complications by direct extension, and in the complication by metastasis, namely, arthritis. In recurrent urethritis, vaccine-therapy is infinitely superior to chemo-therapy. In most cases a combination of both is better still.

GONORRHŒA.

(a) *Acute Urethritis* (*first attack*). Inject intramuscularly 0·5 ccm. colloidal palladium (pallamine) on the first and fifth days, and 0·5 ccm. trimine[*] on the twelfth and nineteenth days, or three injections of pallamine at weekly intervals. With alkalies and aperients internally and irrigations of potassium permanganate ($\frac{1}{1000}$) or trypoflavine ($\frac{1}{4000}$), if the staining properties will not betray the patient, this treatment will clear up many cases, and in all will prevent painful micturition, chordee, etc. If the discharge still continues after the course has been completed, or should a prostatic abscess or epididymitis supervene, either after or while the course is in progress, three intramuscular injections of detoxicated vaccine (2,500^6, 2,500^6, 5,000^6) should be prescribed every second day. If, ten days later, the condition is still acute or subacute, 0·5 ccm. and 1·0 ccm. of trimine should be injected at weekly intervals. If the condition is chronic, 1·5 ccm. intramine should be injected and followed a week later by 1·5 ccm. trimine. A complication will only arise in those cases which presented an acute posterior urethritis when first seen. Taking all cases, I have known a complication to occur only in seven cases since March, 1919. If all cases were put under treatment with pallamine while the disease was anterior, gonorrhœa would never cause more inconvenience than a mild cold in the head.

(b) *Acute and Sub-acute Recurrent Urethritis.* A beginner may find it difficult to differentiate a recurrent urethritis from a second attack, if the discharge happens to be profuse. If he remembers that in over 90 per cent. of patients who develop a discharge, having had gonorrhœa before, that it is a recurrence and not a second attack, he will not go far wrong. If he cannot make up his mind and the discharge is profuse, the case may be treated as a first attack, but in most cases it is wiser to prescribe the three injections of vaccine, and wait 10 to 14 days before proceeding further. When the patient is examined after this interval, particular attention should be paid to the urethra, prostate, and *vesiculæ seminales*. If the lesion affecting one or more of these organs is subacute, two injections of trimine (0·5 ccm., 1·0 ccm.), followed by one or two of intramine (1·5 ccm.), and another of trimine (1·5 ccm.) should be prescribed at weekly intervals. If the lesion is chronic, three injections of intramine, each of 1·5 ccm., followed by one of trimine (1·5 ccm.) should be the course. In both cases, colloidal iodine should be prescribed internally, and the necessary massage and dilatation undertaken.

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[*] Trimine is an improved form of colloidal manganese containing colloidal iron and a trace of colloidal zinc in addition.

(c) *Complication by Direct Extension, including Gonorrhœal Ophthalmia.* Three injections of trimine (0·5 ccm., 1·0 ccm., and 1·5 ccm.) should be prescribed with three days' interval between each. After this a course of vaccines followed by trimine and intramine if the condition is still subacute, or by intramine and trimine if it is still chronic, may be ordered; but, if there is much fear of fibrous tissue forming, intramine should be resorted to as soon as possible. If the case is subacute when first seen, vaccine-therapy should precede chemo-therapy, while if it is chronic three injections of intramine, followed or not by one of trimine, will be all that is necessary.

(d) *Gonorrhœal Rheumatism and Arthritis.* There is hardly any condition met with in medicine which responds so quickly to treatment as gonococcal arthritis does to intramine. However acute the case may be, 1·5 ccm. of intramine should be injected intramuscularly at once, followed by the same dose on the third day, and by 2·5 ccm. a week later.

This course may or may not have to be repeated.

The action of intramine may be enhanced in an acute case by an intravenous injection of colloidal iodine (100·0 ccm.) the day before the first injection, or internally (ʒ iij *ter in die*) in a subacute case for a fortnight before. Colloidal iodine oil should be used locally, and the joint or joints should be moved and massaged as early as possible, to prevent the formation of adhesions. A severe case of gonorrhœal arthritis would never be met with, if an injection of intramine was prescribed the moment the patient experienced any trouble. In this complication vaccines are not called for. To those who wish to try these drugs, let me issue the advice to treat each case individually, and not to put 100 consecutive cases on pallamine, another 100 on trimine, etc., for the latter course can lead only to unsatisfactory results.

VARIA.

As chemo-therapy increases the oxidizing and reducing actions of the protein particles according as metals or non-metals are used, and as these two actions are the combative methods employed by the protein particles to overcome any enemy, be it a parasite or a poison, it stands to reason that the term specificity cannot be applied to the action of any chemo-therapeutic agent. In other words, the chemo-therapeutic drugs mentioned in this article have an application which extends far beyond venereal diseases, as will be seen in a recent article published in the *Prescriber*.[4]

Trimine has an almost magical effect on such lesions as boils, carbuncles, erysipelas, and any very acute septic dermatitis; it will clear up the lymphangitis following a whitlow, and prevent the septicæmia which is so apt to follow. Trimine will cause the disappearance of deep septic lesions, and will prevent a fatal termination of puerperal septicæmia, as I have been able to prove in five instances, but it must be prescribed before the patient is moribund, and therefore is not to be looked upon as a last resort. Colloidal manganese was the only drug which successfully alleviated those suffering from mustard-gas poisoning,[5] and combined with colloidal iodine and intramine it is beneficial in tuberculous lesions.

Colloidal iodine and intramine are the best antidotes for metallic poisoning, they are the only remedies which will cause the rapid disappearance of seborrhœic conditions which are so apt to follow an increased "acidness" of the blood, they are invaluable in rheumatism, whatever be the cause thereof, and are far and away the best treatment for *Acne vulgaris* and *Acne rosacea.*[6] Intramine is also an excellent cleanser and stimulator to chronic sores applied locally and injected intramuscularly.

This work has naturally driven me to consider the life-history of cells, especially the host's protective cells and those undergoing malignant change. Although this is fully dealt with in Part II. of my *Biology and Treatment of Venereal Diseases,*[7] it may be of interest to state here that the lymphocyte is the most important protective cell. Some at least of the protein particles circulating in the serum have their origin in the protoplasm of the lymphocytes manufactured in the lymphatic glands, spleen, and elsewhere. The plasma cell is only a modified lymphocyte, and the protein particles constituting its protoplasm attack the enemy locally in the same way as the protein particles circulating in the serum do generally. The polymorphonuclear leucocyte is of very minor importance, and probably does no more than remove the dead bodies of the parasites killed by the above-mentioned protein particles. If any cell capable of division—note that the polymorphonuclear leucocyte, which has been regarded as our saviour cell is not so constituted, while the true protective cells are—is stimulated or exerts too great an effort to vanquish the foe it is combating, its protein particles undergo certain changes, which result in the cell becoming foreign to those around. In other words, a state of autogenous parasitism is produced. As I have found that such changes are alterations in the colloidal state, which do not fundamentally differ from those witnessed in the immunity reactions *in vitro*, it follows that there is something common to immunology, chemo-therapy, anaphylaxis, and malignant disease.[8]

FINALE.

If what I have stated is correct, the application of colloidal chemistry, which is, after all, the basis of life, to medicine should enable us to discard the illogical theories and the cumbrous terminology consequent on them which have hampered instead of having aided advance. We can retain the things of practical value which have emanated from such theories, but the lines above have shown that none of the tests upon which so much reliance has been based can be considered more than as adjuncts to clinical work. What it actually amounts to is, that most of us have to be born again, unbiassed by what we knew, before we can enter the kingdom of clinical medicine—the goal at which every medical man should aim, for the path of clinical experience is the safest to tread.

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Intramine Dermatitis.

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NO case of a dermatitis following the administration of "intramine" having come to my notice, the following note may be of interest :—

Sapper A. J., 29 years of age, was admitted with the following history. Three months previously he had been admitted to an Infectious Diseases Hospital with the provisional diagnosis of measles. There, syphilis was diagnosed, his Wassermann reaction giving a complete positive result. 0·30 grm. "salvarsan" was forthwith administered intravenously, and a severe "606" dermatitis followed. This went through all its stages, and when exfoliation was complete, and his general condition sufficiently recovered, he was transferred to the Scottish Command Central Venereal Hospital, where he came under my care.

On admission, he had a general adenitis, a well-marked leucodermia on both sides of the neck and on the right and left anterior axillary folds. There was a large eczematous syphilide on the inner surface of the right knee and over the front of the left tibia. Besides a darkly coloured atrophic condition of all finger nails of both hands, he had no other lesion. He was well nourished, and showed no other trace of the severe time he had just come through. His urine was albumen-free. No reliable history of any primary sore could be obtained, and no definite scar was anywhere visible.

From his previous history it seemed clear that he had a very marked susceptibility to "606." As a possible prophylactic, or rather, perhaps, as an endeavour to "acclimatize" him to a greater toleration of the more irritant drug, I proposed giving him a short course of "intramine" intramuscularly before proceeding with any attempt to recommence "606" medication. I accordingly administered 2·50 c.c. "intramine" intramuscularly. Within 24 hours a very close-set, small papulo-vesicular eruption appeared on the flexor aspect of both forearms, shoulder girdle and over both ears. This quickly appeared at the apex of the anal fold, on the inner side of both thighs, and the inner side of the left knee. The eruption over both ears rapidly became vesico-bullous in character, and extended slowly across the forehead without meeting in the mid line. The eyelids were also involved. Next to be affected were the backs and palms of both hands, the eruption here being also vesico-bullous, but the bullæ were smaller, being little more than pin-head in size. The patient had had no iodides whatever.

There was no rise of temperature, no sickness, no diarrhœa, no headache, and his pulse was normal. There was practically no skin irritation, except over the ears. These were treated with applications of calamine cream, and a dressing applied to avoid friction. Otherwise, except for careful dieting, rest in bed and warmth, the condition received no special treatment.

In 21 days the rash had completely cleared, having caused the patient very little inconvenience. The condition was most persistent on the palms of the hands, and a few tiny vesicles on the eyelids caused some annoyance. The patient, however, was in no way perturbed, and merely found being confined to bed very irksome.

Three weeks later I tempted him with 0·15 grm. "novarsenobillon" intravenously. He definitely refused an intramuscular or deep-subcutaneous administration. He received no mercury. There was a rigor four hours after the injection, headache, and a considerable rise in temperature. Next day the face was puffy and swollen, and a severe labial herpes was present. An exfoliative dermatitis followed, and went through the usual stages. In five weeks' time the skin condition was normal. His Wassermann reaction gave an incomplete positive result, and he was returned to his unit with a recommendation for prolonged mercurial treatment, further attempts at arsenical medication being considered inadvisable.

It would seem that in this case the susceptibility of the skin to the action of drugs was very marked, and that he was constitutionally prone to dermatoses.

On the other hand, he may, possibly, have had an idiosyncrasy to one particular group or radical. He had three consecutive dermatoses, one due to "606," one to "intramine," and a third to "novarsenobillon," each following an initial dose. "Intramine" is di-ortho-amino-thio-benzene, and it may be that the common radical was the causative agent in all three cases, namely, the amino or benzene groups. Whether he subsequently showed a similar susceptibility to mercury or iodides, I was unable to ascertain.

Four Cases of Foreign Body in the Œsophagus.

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THE following four cases are reported as illustrative of several practical points in the technique of œsophagoscopic instrumentation. All of them presented themselves within a period of a month; cases 2 and 3 within a few hours of each other. Before describing the actual cases, it may be well to recall the fundamental differences between the older patterns of endoscopic tubes, in which the illumination is at the proximal end, of which Brüning's may be regarded as a prototype, and those patterns the illumination of which is distal, such as Mosher's.

In the Brüning type of apparatus, the lamp is fixed several inches from the proximal end of the tube, down which the light is thrown by a mirror at 45°. Instrumentation is only possible either through a small slit in the mirror, or by displacing it; the available space is very small.

Some of the light is reflected from the walls of the tube, and this reduces visibility at the far end. Moreover, much of the light is lost in its passage, and obviously this loss increases as the tube is passed farther down the gullet. But the lamp is of sufficient size and strength not to be excessively fragile; and being not only outside the patient's body, but also out of the direct line of the tube, is never fogged by condensed moisture from the patient's breath, nor by mucus, saliva, or blood. The only ocular part of the appliance which can be thus dimmed is the mirror, which is in the direct line of fire. But the mirror is protected from rapid fogging by the breath by previous warming, and in any case can be rapidly cleaned without removing the lamp.

Contrasted with this, the Mosher type of tube is illuminated by a lamp placed within three-quarters of an inch of its distal end. The lumen is in profound darkness, so that the distal visibility, where all the light falls on the area under examination, is brilliant; nor is there any loss of light in using an increased length of tube. The proximal end is freely open and unhampered, giving ample room for instrumentation; but to make this possible the lamp is necessarily of extreme smallness and fragility; it is more likely to fuse than is the heavier Brüning type, and to require removal and cleansing when fogged. Since it has to contain the lamp, the tube cannot be made in such small sizes as the Brüning.

CASE 1.—A boy, aged 6, was admitted on May 29, 1919, with a history of having swallowed a halfpenny a few hours previously. On the fluorescent screen the coin was seen to lie in the coronal plane opposite the fourth dorsal vertebra. After a preliminary injection of atropine sulph. gr. $\frac{1}{75}$ and a general anæsthetic (C.E.), Brüning's tube was passed, and the coin easily seen.

The usual instrumental outfit includes two pairs of forceps, extensible by springs; these springs broke, first in one pair of forceps, and then in the other, as soon as touched; they had become eroded with rust through lying unused for some time.

The only other instrument available at the moment was a long blunt hook. This could be passed beyond the coin; but when traction was applied, repeatedly slipped along its circumference and became disengaged. The patient was returned to the ward.

Forty-eight hours later a second skiagram showed that the coin had passed further down the gullet. Through Mosher's tube it was easily seen to be lying in the diaphragmatic hiatus, where it appeared to be firmly held. Gastric juice welled up out of the stomach on each side of the coin, which was readily extracted with Irwin Moore's forceps.

Uncomplicated recovery.

CASE 2.—On June 7, 1919, a girl of 18 was referred to me from outpatients on account of having swallowed an ear-ring four days previously. During this time she had not taken the trouble to watch her fæces for the foreign body. Skiagraphy was negative; but the ear-ring, being a flimsy thing of gold wire, might not have shown up against the dense bone of a transverse process or a rib-head.

On swallowing, pain was felt at a point deep to the sternal head of the left clavicle; which point was also tender to deep palpitation.

Endoscopy with Mosher's tube was instituted at once. No foreign body was seen. But at a point exactly corresponding to the patient's sensations there showed a thin wavy red line, about 1 cm. long. This was not blood-clot, for it was not removable by swabbing. Examination was continued down to the cardia; and the gullet once more slowly scrutinized on the return journey of the tube. The same red line was seen again; presumably a scratch, produced by the ear-ring during its sojourn. The patient's sensations gradually disappeared, and the foreign body had probably been passed per rectum.

Case 3.—A boy, aged 4, was referred from outpatients on June 6, 1919, within a few hours of Case 2. Some hours previously he had swallowed a halfpenny, since when he had been unable to swallow anything else. On the X-ray screen the coin was seen lying deep to the manubrium; and although he vomited soon after admission, it was not ejected.

The child was undersized for his age: weight 2 stone 5 lbs., height 3 ft. 2 ins. He was much too small for the safe passage of a Mosher's tube, or of the largest in Brüning's set.

Brüning's No. 2 tube (diameter, ½ inch) was passed, and the coin instantly removed with Irwin Moore's forceps, the whole manœuvre occupying less than a minute. Half an hour later he was sitting up in bed, playing with toys, and was made to lie down again.

No complications supervened, and he was discharged on the sixth day.

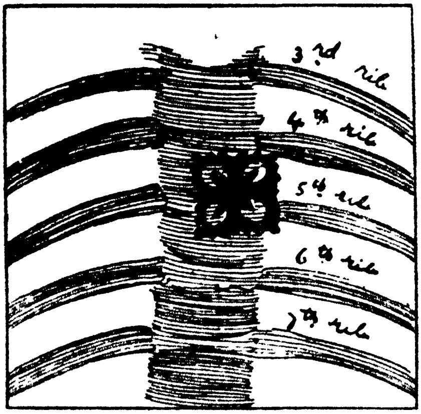
Two days later he was brought back to Hospital, crying and in obvious discomfort. There were no physical signs, and, before I could arrive, his mother elected to take him home again, where, five hours later, he collapsed and died suddenly.

Autopsy (at the Coroner's court, the patient having died at home).—A perforation of the œsophagus opposite the cricoid on the left side, leading to an abscess, which extended to the anterior mediastinum and involved the thyroid gland. The abscess cavity contained a greenish substance. This was unfortunately not preserved for examination, but was described by the pathologist as "resembling cabbage"—which it may have been. The sinus opened out of the gullet several inches above the point where the screen had shown the coin, and from where this had been extracted. The probable explanation is that, as commonly, the foreign body had been impacted behind the cricoid, and, while there, had given rise to an ulcer, which subsequently perforated.

Case 4.—A boy, aged 4½, was admitted to hospital on June 19,

1919. Three days earlier he had swallowed the buckle of a velvet head-band belonging to his mother.

In the skiagram the buckle strongly resembled a metal star from a military epaulette; after removal it was found to be an imitation diamond buckle, square, and ⅝ inch in each diameter.



The skiagram did not include the clavicle, but it extended to the 2nd lumbar vertebra below; the level of the foreign body could therefore be seen by counting the ribs from below upwards. In this drawing of the skiagram the lower part has been omitted.

The first skiagram showed the buckle, as usual, in the coronal plane, opposite vertebra D_2. A second skiagram proved that it had descended to the level of the intervertebral disc between D_4 and D_5.

Atropine sulphate gr. $\frac{1}{100}$ was given, preliminary to chloroform anæsthesia. The second largest sized Brüning's tube (diameter, ½ inch) was slowly passed, and the gullet minutely examined from cricoid to cardia three times. No foreign body could be seen, nor felt with a long bougie or probe. The only abnormality noted was congestion of the mucosa in the lower half of the gullet. The next day the search was repeated, with the same tube and the same failure.

The prospect was very grave. The child was a puny Eurasian, only 3 feet 2 inches tall and weighing but 2 stone 1¼ lbs., and I did not dare use the largest Brüning's tube or Mosher's apparatus. Moreover, situated as it was, in the middle segment of the œsophagus, the buckle was not conveniently accessible, either from above, by œsophagotomy, or from below, by retrograde extraction, through the stomach. It was now four days since the accident; and in the skiagram the buckle could be seen to have four sharp angles. (The mother's idea that it bore two hooks on the posterior surface proved, later, to be imaginary). To move the patient to the X-ray room and search for the foreign body, aided by the screen, would have taken too long, having regard to the child's collapsed condition under this second anæsthetic. With a gloomy anticipation of failure, the coin-catcher was passed, unaided by sight. At the third attempt the buckle was extracted.

Recovery was uneventful.

CONCLUSIONS.

Apart from the technical merits and demerits (briefly alluded to above) of various types of endoscopic apparatus, a few points, some of them well recognized, find emphasis in this small series of cases :—

1. Frequently a patient insists that a foreign body is still in the gullet, when as a fact it has passed on, as in Case 2; the resulting trauma accounting for the persistent sensation. More than this, a patient will vehemently insist that a foreign body is impacted when there has never been one; the classical instance of this being, of course, globus hystericus. Indeed, the history is often just as unreliable as when we are dealing with the question of a foreign body in nose or ear, but unreliable in a converse sense; for in the latter the patient generally denies any suggestion of a foreign body.

2. Among hospital patients swallowed coins are usually coppers. A penny is too large to travel far in a child's gullet. Considering, therefore, only halfpennies and farthings, it may be said that the latter will, as a rule, eventually pass on into the stomach of any child of average size, aged five years or more, or be regurgitated into the mouth; whereas a halfpenny usually becomes impacted.

3. For extraction, hooks are often useless; with the possible exception of a swallowed ring, into which a hook may be engaged, it tends to slip off the foreign body; a hook, also, is likely to scratch the mucosa, causing subsequent perforation. Forceps containing complicated and delicate parts (such as spiral springs) which may rust and break, are not good; a strong, simple forceps, such as that of Irwin Moore, is the

proper implement.

4. The well-known fact that any foreign body is likely to be impacted behind the cricoid must be remembered, even if the former is eventually removed from a lower level in the œsophagus; for, during its probable sojourn in the post-cricoid region it may ulcerate and perforate. Thus, in Case 3, if the first skiagram had not shown the coin behind the cricoid plate, the reporter would have been in doubt whether the ultimate fatality had not been due to his instrumentation.

5. In the event of fatal ulceration and perforation, death may occur very late after an interim of perfect health. As a rule, a patient may be regarded as safe after a week; but Case 3, perfectly well until the ninth day, died of a massive abscess in the cervical fascial planes.

6. In Case 4 the final success with the coin-catcher, after repeated failure with the œsophagoscope, reminds one of the small boy catching many fish with a bent pin, while experienced anglers, using rod and line, are having no success. Evidently the humble coin-catcher (although no doubt used preferably in conjunction with the fluorescent screen) should not be relegated to the museum of surgical antiquities.

Sycosis Barbæ.

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IT is not always realized that the ætiology of the sycoses is complex. Many of the text-books, containing articles on this important subject, content themselves with a brief *résumé* of its bacteriology, and a discussion of the relative importance of the strepto- and staphylococcus as the primary instigators of the infection. Such discussions appear to the present writer to be of academic interest only, for the biological means of coping with the infection of hair follicles by vaccines—whether autogenous or from a mixed stock—are notoriously inefficient to effect a cure by themselves.

The various types of the infection, and the diseases which can more or less closely imitate them, will be discussed under the following heads, the remarks on treatment being mostly confined to the common variety which is best known under the name of "barber's rash":—

(1) Seborrhœic sycosis.
(2) Barber's rash. True coccogenic sycosis.
(3) Tinea sycosis.
(4) Lupoid and syphilitic sycosis.
(5) Miscellaneous infections which may superficially simulate the sycoses.

(1) *Seborrhœic sycosis* is always part of a more or less generalized infection of the hairy regions. It follows, therefore, that it is essential in any case of sycosis to ascertain if the infection of the

beard region is of long standing, or gives a history of frequent relapses. It is of prime importance to establish or negative the coincidence of seborrhœic involvement of the scalp, or eyebrows, or the presence of chronic marginal blepharitis, which is usually proof positive of the existence of a "seborrhœic diathesis"[1] from childhood or infancy.

If these areas are found to be also involved, the treatment will be exceedingly difficult, and a guarded prognosis should be given. This type of sycosis provides the bulk of chronic and incurable cases.

Relapse is very common after X-ray epilation, and although such a desperate cure should never be aimed at, areas which by repeated radiotherapy at more than one institution have become permanently bald, do not again become infected.

One such patient, in whom the normal skin of one side of the face and chin has been replaced by the atrophic scarring and pigmentation of excessive radiotherapy, much prefers the disfigurement to the disease, and never ceases to importune his medical advisor for a similar "cure" of the side still affected.

Before considering in detail the type of sycosis, which it is the main purpose of this contribution to discuss, a brief reference must be made to that variety which is dependent on a chronic infection of the nose or oral cavity. In the majority of these the folliculitis is limited to, or most obvious on, the upper lip, and is often associated with chronic thickening due to lymphangitis.

The diagnosis is usually obvious at a glance, or from the history of persistent nasal catarrh, and the local condition is not amenable to treatment until measures directed against the intra-nasal factor have been undertaken.

In this connection, too, a dentist may have to be consulted with regard to the existence of pyorrhœa or sepsis of the gums and teeth. A recent case of the writer's, in which the infection had remained localized to the central depression between the lower lip and the chin for three months, and had resisted all local applications, cleared up at once without any local treatment after scaling and cleansing the central and lateral incisor teeth of the lower jaw.

(2) *True coccogenic sycosis*, familiarly termed "Barber's rash," is always limited to the beard region. It is a disease which is directly caused by shaving, and is thus confined to the male sex at the age at which this tribute to convention is general.

Clinical observation will establish the fact that it is more common in men with stiff or coarse beards than in those endowed with hair of finer texture.

The pseudonym of "Barber's rash," by which the condition is popularly known, implies the transmission of infection from dirty utensils in the barber's parlour. The imputation thus conveyed is a very serious one, and in the large majority of cases wholly undeserved. The present writer is not in a position to give actual figures, but he has made it his practice to enquire into the source of infection in every case of coccogenic sycosis seen at the Great Northern Hospital since November last, and in not one single instance was the accusation made. It can be stated with complete confidence that the preponderance of cases occurs in patients who are in the habit of shaving themselves, and that the commonest cause of the malady is *not*, as has been hitherto tacitly assumed, an infection from without, but is the result, in the first instance, of the irritation produced by a blunt razor, whether of the simple variety or of the modern "safety" pattern. The edge of a razor, even when it is sharp, is toothed and jagged, as can easily be proved by examination with a low-power microscope. If such an instrument is placed in a hasty or unskilful hand, or in the powerful leverage of a safety holder, it is not difficult to imagine the number and nature of the minute, and daily repeated excoriations of the epidermis.

That such abrasions should occasionally become infected is not surprising. Both staphylococci and streptococci are present on the normal skin, and become potent and virulent on one which is irritated. Actual cuts with a razor blade are seen at once, and therefore avoided at the next shave. Not so the microscopic abrasion, which is flat enough to lie in the superficial plexus of lymphatic, and often heralds its presence not so much *ad oculos*, as by the sensation of itching it produces. This sensation, which the present writer has himself experienced, is actually relieved momentarily by the passage of the razor, or by rubbing or scratching, which, of themselves, further irritate and spread the infection.

Whether the intermediate skin or the hair follicle is primarily infected, does not seem to be of any great importance. The important fact is that a septic focus has been established from which infection radiates. On the third—sometimes even on the second day—the result of this infection becomes apparent to the eye. Individual hair follicles are surrounded by a pink areola of inflammation, and here and there are papules or vesico-papules from which a quickly drying, clear, yellow serum exudes. Bacteriological examination at this stage will convince the majority of investigators that the streptococcus is the responsible agent. On the morrow, when the clear serum has become opaque or distinctly purulent, the staphylococcus will be blamed, and very probably inoculated on agar culture mediums for the purposes of a vaccine.

Active local measures for cure will be in full swing by this time. As the infection has been assigned—and correctly so—to the cocci, antiseptic remedies, well known for their powerful bactericidal effects in impetigo contagiosa and allied infections, will have been freely applied.

Paramount and favourite among these is the official P.B., unguentum hydrarg. ammon. dil. Every chemist's assistant knows that it is an unfailing remedy in impetigo and ringworm of the glabrous skin, and few and lucky the patients who escape its local inunction, and the direct aggravation of their complaint by its unskilled application. Many other ointments and lotions are prescribed, but nearly all of them have a grave fault in common—*they are too strong.* The more important of the underlying ætiological factors—that of irritation—is thus ignored, or even *added to,* with clinical results that are uniformly disappointing.

A week or more is usually wasted in unsuccessful attempts to find a specific remedy, and during this time the patient has, as a rule, had at least one dose of a polyvalent vaccine.

Such is the common history attaching to most cases of the infection. On examination, it will be found that a very large number of hair follicles are involved, and there is usually a considerable catarrh or dermatitis of the intervening skin, partly as a result of the irritation produced by sepsis, partly owing to the application of unsuitable remedies.

The problem of therapy must be attacked according to the stage in which the patient is seen. In the first stage, which may for descriptive purposes be termed the stage of irritation, shaving with a sharp razor and a light hand in the downward direction only may be allowed. It is, perhaps, needless to commend also the use of a good, fatted, non-irritating shaving cream or soap, which should be lathered gently into the face with hot water for at least five minutes before the actual operation is begun. The second desideratum (after removal of the soap), is the immediate application of a soothing lotion, of which the simplest is one containing a drachm of prepared calamine to an ounce of lime water, to which, especially in hot weather, a little eau de cologne or pure spirit may be added with benefit.

This lotion should be mopped on to the whole face and not rubbed in. It should be gently dried off with a clean face towel. A careful watch must be kept for the development of the second stage in which irritation and infection coexist. Shaving should be forbidden for at least 48 hours and on this account alone, if not because of the necessity of frequently repeating the local applications, the patient will be quite ready to obey the injunction to stay in the house for at least a day. The local applications which are efficacious in this stage are mildly antiseptic, and the best are the salts of lead and mercury. If the latter is used, a strength of $\frac{1}{6000}$ of the perchloride should not be exceeded. The most valuable salt, however, in the writer's experience for this stage of the disease is the cyanide of mercury ($\frac{1}{4000}$). This powerful but unirritating antiseptic is very little used in this country, probably on account of its popularity with intending suicides. It can, however, be procured in tablet form, and has little or no oxidizing effect on the nickel or silver plating of surgical instruments. The use of antiseptic lotions in the second stage should be alternated with the alkaline calamine lotion above described, especially if the skin shows signs of becoming macerated or sodden.

On no account should ointments of any kind be applied in this stage. Their medicinal contents must needs be irritating if efficiently bactericidal, and the greasy nature of the bases employed tends to maceration of the œdematous epidermis, and spread of the infection from one hair follicle to another, under cover of a greasy protecting film. This also tends to dam back the issuing secretions and interfere with the efficient cooling of the inflamed surfaces. Whether such is the true explanation of the action of an ointment or not, is immaterial, "Experientia docebit."

The third and indeterminate stage is one the necessity of treatment for which should seldom arise, for efficient therapy should always be successful in one or other of the preceding stages.

Once these are passed, and the micro-organisms have thoroughly established themselves in the hair follicles and their intricate connections, effective control of the disease will tax the utmost resources of the therapeutist. He is then faced with the same problem that confronts the surgeon who is attempting to maintain proper drainage of an intricate abscess cavity. The case cannot be dealt with effectively until such drainage is effected by loosening and detachment of the hair from its sheath. In acute cases this generally takes place by suppuration and necrosis in the neighbourhood of Henlé's layer. The hair then either comes away naturally, or can easily be removed with forceps. Sometimes the process can be accelerated by fomentations, though care is necessary here in order that by too prolonged a contact (especially with boric lint) the poultice does not become an incubation chamber. Plain white lint or gauze soaked in hot $\frac{1}{4000}$ mercuric cyanide is probably the most valuable agent for the purpose. In cases which have become chronic over three or four months, X-ray epilation is the best treatment. **The X-rays should never be used in acutely inflamed *septic* cases. All secondary dermatitis must first be allayed with calamine, lead, or other soothing applications, *for several days* before a full epilation dose is** decided upon.

Some cases with decided dermatitis superadded—a weeping catarrh of the skin—do not seem to respond to local applications of any kind. Such cases it is sometimes permissible to expose to very minute doses ($\frac{1}{5}$ to $\frac{1}{8}$ pastille) of the rays with good sedative effect. Epilation can then be proceeded with at a later date.

In other types, in which there is a lot of impetiginous crust formation, the use of an oily, mild, alkaline liniment as, *e.g.* :—

Calamin Prep. - - - -	ʒii.
Ol. Olivæ - - - - -	ʒiii
Aqua Calcis - - - -	ad. ℥i

is indicated.

It must be kept continuously applied, and if not quickly successful should be replaced by the occasional application, *for not more than one hour at a time*, of a boric starch poultice, the preparation of which is so admirably described by Norman Walker in his "Introduction to Dermatology."

The writer cannot record any decided success from the use of vaccines, and, for the same reasons that face the surgeon in the case of a partly draining abscess, he has practically abandoned their use in this disease.

During the stage of X-ray epilation, which is always quicker in the case of the infected hairs, the use of cooling or mildly antiseptic lotions or liniments should be continued.

It is only after epilation has occurred and all inflammatory symptoms, whether of septic, medicinal, or radio-reactive origin, have subsided, that the use of ointments is justifiable. Even then, a paste, which is an ointment containing starch, kaolin, or some other absorbent of water, will probably be better tolerated at first. If epilation is complete and the case is not really part of a general seborrhœa, a cure even at this stage is the rule, and can be predicted with a confidence which grows as experience of actual cases is multiplied.

(3) *Tinea sycosis.*—The infection is usually contracted directly or indirectly from horses and dogs. On examining the former animal, the lesions are of a chronic type, and the appearance is not unlike that seen in the common type of human ringworm of the scalp in children. There is considerable loss of hair, and, instead of being glossy to the touch and sight, the coat looks mangy and moth-eaten. Similar appearances are met with in the dog, but in a recent case of the author's, in which a lady had been infected on the face by her Pekinese spaniel, the latter's skin showed severe inflammatory reaction with loss of hair, not unlike the kerion-type of lesion resulting from endothrix infection of the scalp.

When ringworm infects the beard in a man, there is almost invariably a severe inflammatory reaction. The individual lesions, which may be two or more in number, are circular, of a size varying from half-a-crown to the circumference of an egg-cup. The inflammation, which is an expression of local reaction and a stage in the production of immunity, is always accompanied by œdema, which tends to raise each circular plaque considerably above the surface of the skin. This feature is almost invariably present, and of the greatest diagnostic importance.

The hair follicles themselves secrete pus, and the hairs can be epilated without pain as a rule, for they come away very easily. As in kerion of the scalp—and the present writer can see no reason why the focus on the chin should not also be termed kerion—the search for mycelial elements (wrongly termed "spores") in the hairs is commonly unsuccessful. The inflammatory leucocytic reaction has in all probability disintegrated them at this stage.

Culture tests on Sabouraud's agar medium will often prove positive, however, and this ætiological proof, though slow, is always worth doing if there is the slightest doubt in the diagnosis.

As in chronic coccogenic sycosis, the X-ray epilation of the hairs in this infection also has been urged in most text-books, but the writer's experience of this treatment has not been a happy one. It, should be confined, in his opinion, to such rare cases as are tending to become chronic. The resulting reaction after X-rays in such cases is violent in the extreme, and in one case, in which this operation was performed in France, it was undoubtedly responsible for severe aggravation and spread of the condition to the whole face and neck.

Epilation by hand and frequently repeated hot boric fomentations have always sufficed to shorten the course of the disease in the author's hands. Even mild antiseptic lotions are badly tolerated, and shaving must be absolutely forbidden, the beard being kept reasonably short with scissors for the time being.

Immunity is usually established in from six weeks to two months, and this very definite course encourages the hope that it will ultimately be possible to prepare a successful vaccine—the attempts made hitherto with so-called "trichophytins," in Austria and elsewhere, having achieved only partial success.

(4) Lack of space forbids more than a very superficial consideration of lupoid and syphilitic sycoses. The term lupoid sycosis is a misnomer. It is an exceedingly rare affection and differs from coccogenic sycosis, by which it is believed by some to be preceded, in spreading with a raised infiltrated

border, behind which the hair follicles are atrophied and cicatricial. According to Sequeira[2] and others, there is no evidence to support a tuberculous ætiology.

Syphilitic sycosis should offer no difficulty in diagnosis. In the secondary stage, it is inconceivable that the eruption would be limited to the beard, and in the tertiary stage there would be the typical serpiginous edge with gummatous ulceration and scarring, together, probably, with other evidence which would establish the true nature of the affection.

An error could only arise if the lesions were covered with crusts, and would certainly be corrected at the second visit, when these had been removed by starch poultices or other means.

(5) Among the miscellaneous affections which may occasionally be limited to the beard region, but of which mention must suffice, are—

(*a*) *Primary syphilitic chancres*, which may be multiple.

(*b*) *Anthrax* (malignant pustule)—always associated with severe prostration and pyrexia.

(*c*) *Glanders*, in which the same general symptoms occur.

(*d*) Malignant disease, such as *rodent ulcer* and *epithelioma*.

(*e*) *Actinomycosis*, *lupus*, and *lupus erythematosus*, and *sporotrichosis*, all of which might conceivably have to be considered in the diagnosis.

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[2] Sequeira: *Diseases of the Skin*, 2nd edition, pp. 192, 193.

The Varieties and Treatment of Retroversion of the Uterus.

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WHEN examining a woman on account of pelvic symptoms one of the commonest of physical signs discovered is backward displacement of the uterus. The condition is, as a rule, easy to diagnose, for, in many cases, it is unmistakable, but the difficulty arises when the attempt is made to assess the condition as a cause of symptoms.

The number of symptoms which are ascribed to it are very many, and a fine judgement is sometimes necessary to determine whether such are really set up by reason of the uterus being directed backwards instead of forwards. When a retroverted uterus is found with no other readily discoverable physical sign to account for the pain, bleeding, or other trouble, it presents itself as an obvious morbid condition for which treatment can be carried out. It is felt that here is something at any rate which does not conform to the normal of the text-books, and if it is corrected the symptoms may improve.

This is often done by pessary or operation, with the disappointing result that the patient is no better. She may even be worse off, because, by reason of the treatment, her attention has become more focussed on her pelvis, pain thereby becomes magnified, and a vicious circle is established. It is necessary then to be cautious in the selection of cases for treatment by replacement, and it should be only carried out when it is fairly certain that the retrodeviation is the actual cause of the trouble.

The error of attributing too much to backward displacement arises from the former idea that the condition is usually a primary one, and, therefore, responsible in itself for symptoms, but a careful scrutiny of patients will reveal that in the majority the abnormal position is more often incidental to other factors which are present and causative.

In fact, it is seldom that a simple, uncomplicated retroversion without associated conditions such as prolapse of uterus or ovaries, or endometritis, should ever be treated. All the symptoms, except, perhaps, dyspareunia, usually ascribed to retroversion more frequently occur when the uterus is forward, *i.e.*, of all the women examined for any one of the symptoms, many more of the patients will be found with the uterus anteverted than retroverted. In the same manner the uterine physical signs of slight enlargement, softness, tenderness, and immobility are more commonly found in anteverted uteri.

Let us classify the varieties into—

I. Primary, or congenital.
II. Secondary, or acquired.

I. The congenital variety is sometimes met with in young virgins, who are examined for "spasmodic" dysmenorrhœa. The uterus is usually of the pubescent type, *i.e.*, small and acutely bent at the cervico-corporeal junction. It is, however, very freely movable, and not in the least tender. Dysmenorrhœa, as acute, is much more commonly seen with uteri of similar shape and development in the anteverted position. In these cases, it is very probable that the position of the uterus is entirely incidental, and in no way can be the cause of the monthly pain.

II. The six common varieties of the secondary form are as follows:—

(1) Retroversion following labour or abortion:—During the puerperium, two factors may be present which favour malposition—the dorsal position of the patient in bed and abnormal softness of the uterus during involution, due to sepsis, often of a very mild type. If these factors are present after the eighth day, the heavy, bulky, and soft corpus uteri may fall backwards after it has sunk below the support of the sacral promontory, and it will remain backwards unless artificially replaced.

But permanent, also, will be the subinvoluted state of the uterus, with its endometrial thickening and hyperæmic musculature.

Whether these latter changes which cause the symptoms of bleeding and pain are increased by the retroverted position is very doubtful, for we find precisely similar conditions of equal intensity in subinvoluted anteverted uteri. The mechanical view that the broad ligaments are kinked over the sharp edges of the utero-sacral folds cannot be held. How rarely is this seen on viewing the retroverted uterus through a laparotomy wound! The utero-sacral ligaments in these cases are particularly soft and flabby, and quite incapable of causing a sharp kink in the broad ligaments and compressing their veins. Moreover the anastomotic circulation of the uterus is so free that venous impediment would have to be very complete in order to cause real congestion. Further, common experience teaches us that retroverted uteri can undergo complete involution after parturition.

(2) Retroversion is very commonly seen with prolapse of the uterus and vagina, usually in their later

stages. It is probable that backward displacement may occur as the primary condition in the rare form of prolapse known as utero-vaginal, seen in nulliparous women, in which the first element to prolapse is the uterus passing into the upper part of the vagina, without perineal injury and evident cystocele and rectocele. In the ordinary form of prolapse, vagino-uterine, in which the perineal injury is the primary cause of the trouble, retroversion is thought to be secondary to the dragging downwards of the vagina. This is borne out by the observation that the malposition of the uterus is generally seen in the more advanced degrees of prolapse. That it, certainly, has an accelerating effect on prolapse is gathered from the facts: (i) when retroversion has become established the progress of the prolapse is more rapid, and may become complete procidentia; (ii) that operative cure of prolapse, short of restoration of the uterus into the forward position, is not so successful as when the uterus is suspended forwards in addition to the performance of perinæorrhaphy; (iii) so long as the uterus remains forwards, the worst forms of prolapse are seldom seen.

(3) There is a common form of retroversion in which the uterus is not enlarged or the subject of endometritis, but where the uterine supports and pelvic diaphragm are in a slack and flabby condition.

On opening the abdomen the body of the uterus is seen in Douglas's pouch, the round ligaments are thin and elongated, in fact, little more than thin ridges of peritoneum, while the utero-sacral ligaments can scarcely be identified. The uterus is very movable, at a lower level than normal, and swings forwards and backwards without any restraint at all. Ordinarily, if the normal uterus is pressed backwards, the round ligaments are seen to tighten and check the movement, but in this variety everything is flaccid and loose.

In addition, the ovarian supports share in the general slackness, with the result that the ovaries usually lie low down on either side of the uterine body or even underneath it. The infundibulo-pelvic ligaments are frequently bulging with large dilated veins, virtually a condition of varicocele. On lifting the uterus up to the vertical or forward position the ovaries still hang down very low, and may scarcely be raised from the pelvic floor owing to the thin and relaxed state of the ovarian ligaments.

The patient is always a multipara, and frequently has a slack abdominal wall—in fact, the pelvic condition is part and parcel of a general toneless state.

The symptom complex is definite: backache, and "bearing down" pain, due to the constant dragging downwards, but relieved on lying down almost instantly; premenstrual pain, due to ovarian congestion, without menorrhagia; and, particularly dyspareunia of ovarian origin.

In these cases the retroversion is probably not an important item, but indirectly it is a fault on account of the complicating ovarian prolapse. This is the chief symptom-causing factor present, by reason of dyspareunia and the backache caused by the engorged pampiniform veins. It is the ovarian mal-position, then, which chiefly calls for treatment, and this is partly served by anteverting the uterus as a first measure.

(4) A further variety of retroversion is found with precisely opposite anatomical conditions to those just described. Here, on opening the abdomen, it is seen that the uterus is small, and apparently held down backwards by short and tense posterior and broad ligaments. There are no adhesions, and no sign of tubal inflammation, but the uterus will

scarcely come up to the vertical because of the tension of its posterior and lateral attachments, nor can it be raised to the abdominal wound.

The ovaries are small and closely associated with the lateral borders of the uterus, with very little range of mobility, but without real adhesions. With the contraction of the posterior ligaments, the round ligaments are correspondingly thin and atrophic, and are quite useless as a means of support of the uterus against its posterior tether.

Clinically, the patients are generally over 40, and sterile, with a long history of pelvic pain, and particularly dyspareunia. There is neither menorrhagia nor leucorrhœa. On examination, the most prominent physical sign is the retroverted uterus, which is extremely painful when the slightest attempt is made to replace it. The uterus itself if palpated very carefully from the vagina is not really tender, but as soon as any pressure is made on the fundus the increasing tension on the posterior and lateral ligaments thus set up apparently causes very great pain. The condition may be one of congenital hypoplasia, and thus properly fall into the first category described in this paper.

(5) Chronic salpingo-oophoritis is usually associated with retroversion. The uterine position is due to the dragging of the uterus backward by the inflamed and heavy appendages, which themselves drop to the bottom of Douglas's pouch. Adhesions rapidly render the displacement fixed and permanent. In these cases, the position of the uterus is entirely overshadowed by the presence of chronic pelvic peritonitis, and cannot be treated apart from the primary cause.

(6) Finally, the uterus may be pressed down backwards by a superincumbent tumour.

When a patient is examined for repeated abortion it is sometimes found that she has a retroverted uterus, probably slightly enlarged, and she complains of excessive monthly bleeding. There may be no other discoverable cause of abortion such as syphilis, and the conclusion is made that the abortion is due to the mal-position of the uterus.

Now the figures given by Herman clearly show that retroversion is not a cause of abortion, and that below the age of 35 it has little influence on fertility (Herman and Maxwell, *Diseases of Women*, p. 626). Moreover, it is common experience that women having retroverted uteri can easily conceive and carry to full term. It is difficult to see why a uterus, normal in every other respect, will fail to carry merely because it is lying backwards, for it has complete mobility and can readily move forwards and upwards to accommodate the growing ovum. Again, we find the incarcerated retroverted gravid uterus usually obstinately maintains its contents in spite of really gross embarrassment, jammed down, as it is, behind the sacral promontory. In fact, in rare cases, a uterus so placed, and unable to right itself, may even continue to develop by enormous sacculation of its anterior wall.

Therefore, there seems to be no inherent reason for abortion lying in mere retroversion, and when the two occur together it is necessary to look further for the cause. It will be noted, on careful examination, that the uterus in such patients complaining of repeated abortion is slightly enlarged and tender, and, if curetted, will yield thickened scrapings. Endometritis and less commonly chronic metritis are the causes of the trouble, and must be recognized as being far more important than the position of the uterus.

Sterility is not due to backward displacement alone, except, according to Herman (*see* above), over the age of 35, when it does operate to a very slight extent. It will, however, occur if there is ovarian prolapse causing severe dyspareunia, and may also be present if there are inflammatory changes in the uterus.

TREATMENT.

From what has been discussed hitherto, it is clear that most patients exhibiting retroversion require treatment for the associated conditions rather than the displacement, and further that simple replacement of the uterus will do little or nothing to relieve symptoms. Let us consider, *seriatim*, the varieties just enumerated from the point of view of treatment.

I. Congenital retroversion should not be replaced. It comes under our notice during the examination of a patient complaining of spasmodic dysmenorrhœa or sterility, but no correction of this uterus will cure either symptom. If the attempt is made to bring it forward by the bimanual method it will be found that the uterine tissue at the cervico-corporeal junction is rigid, effectually preventing any permanent replacement because there can be no undoing of the retroflexion, the uterus is rotated as a whole, and when the fingers are removed the organ immediately slips back to its former position. The proper treatment of these cases is that for spasmodic dysmenorrhœa, such as suitable drugs to relieve pain or dilatation of the cervix.

II.—(1) That form of retroversion coming under our notice for menorrhagia and pelvic pain following abortion or parturition, should be treated for endometritis, or, in later years, chronic metritis, which are the real causes of the symptoms. In all but the slightest cases it is necessary to curette the uterus, after which it should be swabbed out with 10 per cent. formalin solution, care being taken that the fluid does not drop into the vagina or on to the skin.

At the same time opportunity should be taken of the anæsthesia to replace the uterus manually. The added tone which curetting imparts to these uteri will probably be sufficient to assist the organ to remain in the anteverted position. During the ensuing fortnight the patient should have a mixture of ergot and hydrastis, and before she gets up the position of the uterus should be again examined and if necessary replaced bimanually with the temporary insertion of a pessary for a month or so. This is done to prevent subsequent prolapse of the ovaries, which would give rise to a further train of symptoms. A final examination is made a few days after the ring has been inserted to see that it is efficient in maintaining the uterus forward. If it is found that the pessary has already allowed the uterus to drop backwards, it should be removed as it can serve no useful purpose, and the uterus may remain as it is.

The exceptional cases are those in which there is noted a tendency for the ovaries to prolapse, or where the uterus itself is in a state of incipient prolapse. Here it is necessary to regard the mal-position as of importance, and correct it by a suitable operation such as Gilliam's.

(2) Retroversion as a part of prolapse requires treatment. In the slighter degrees of vaginal prolapse when the cervix does not appear at the vulva, the retroversion may be adequately treated by a temporary pessary after an operation for repair of the perinæum. If, however, the uterus appears at the vulva, and is definitely enlarged, the perineal operation should be preceded by curetting and followed by Gilliam's round ligament suspension. Perinæorrhaphy is not enough by itself. The uterus is left lying in the same axis as the vagina, and there is nothing to prevent it falling into the vagina in the form of a utero-vaginal prolapse. Replacement manually followed by a ring is useless because the cervix is elongated, rendering recurrence of backward

displacement certain. It is therefore necessary to maintain the uterus permanently forward by abdominal suspension, or, less commonly, fixation. This measure restores the perpendicularity of the uterine and vaginal axes, prevents the telescoping of the uterus into the vagina, and in the majority of the cases completes the cure. It is rarely necessary to amputate the cervix, for there is evidence recently brought forward to show that after the cure of prolapse the length of the cervix greatly diminishes.

(3) Retroversion associated with relaxed ligaments and a flabby pelvic floor may need correction on account of prolapsed ovaries. In slight cases, in which the symptom is chiefly dragging pain and aching on standing, a ring pessary will frequently enable the patient to avoid operation, for a pessary is not always contra-indicated when the ovaries are prolapsed. If it succeeds in carrying the uterus forward, the ovaries may be lifted up with it, and will thus be out of reach of pressure by the ring, and sometimes the ovaries are elevated on the ring without pain being caused.

But if the pessary fails to lift the uterus forwards, it usually causes pain because the congested and tender ovary lies between the ring and the fundus. Should the ring fail, or especially if dyspareunia is a prominent symptom, Gilliam's or a fixation operation will be necessary, followed by a long rest in bed.

After the round-ligament-suspension part of the operation is done, the position of the ovaries should be carefully examined. Sometimes they are carried well up by the anteverted uterus, but generally they will require suspension by a pleating stitch run along their ligaments. This method is very successful, and causes them to lie well up against the uterine cornua.

(4) The unusual form of retroversion in which the

uterus is held down by contracted posterior ligaments can only be treated properly by hysterectomy. It usually occurs in sterile women over 40, and is the cause of much suffering, especially severe dyspareunia. Suspension is impossible owing to the feeble round ligaments and the difficulty of pulling the uterus up. Subtotal hysterectomy is the only means of curing the patient.

(5) Associated with chronic salpingo-oophoritis, rectification of the retroversion is unimportant except after the removal of tubes and ovaries, if that operation is being done. When the inflammatory tissue has been removed, attention must be given to the uterus. It is nearly always retroverted or else threatens to fall backwards (on account of the contraction of the broad ligaments following ligature of the stumps), it is frequently enlarged, and has a raw posterior surface.

If it is in a state of chronic metritis with much enlargement, it is better to remove it with the tubes and ovaries, but if its size is fairly normal and the patient is young, and especially if the appendages on one side have been conserved, it should then be suspended forwards in order to prevent the raw surface becoming adherent to the rectum.

To sum up, retroversion requires treatment when it is a cause, direct or indirect, of dyspareunia, or associated with prolapse of the uterus, vagina, or ovaries, and in some cases after the removal of the tubes and ovaries for chronic inflammation.

In other cases, correction of the displacement is a useful adjunct to treatment such as curetting.

An Undiagnosed Disease probably of Infectious Nature.

By C. F. ORR WHITE, B.A., M.R.C.S., L.R.C.P.
Late Resident Medical Officer, Hertford County Hospital, and Captain R.A.M.C.

WHILE I was on duty at King George's War and Station Hospital, Poona, India, I was temporarily put in charge of the skin department on August 30, 1919. The previous day four cases were transferred from Ahmednagar to King George's War Hospital, Poona, which had been seen by various members of the staff of the latter, but presented certain difficulties in diagnosis. All of them came from a unit of the East Yorks, which had freshly arrived in India. Two of these cases had been tattooed recently (some three days previously), and the new tattoo marks showed signs of local inflammation, which subsided without any sign of suppuration. The remaining two cases had not been tattooed recently. All four cases presented similar symptoms. There was slight fever—in no case exceeding 100° F.; by the next day the fever had disappeared. The men did not then complain of any definite symptom and ate and slept well.

In each case a rash was present with circular flat papules, which did not entirely fade on pressure. These varied in size from a pea to a two anna silver piece (smaller than a threepenny bit), were most pronounced on the wrist and forearms, and were beginning to appear on the chest, thighs, and legs.

It was suggested that bug bites (just then very frequent), combined with a dermatitis, might be the cause. These cases corresponded to no condition resembling anything I had seen during three years in India. The rashes were fading rapidly when on September 2, 1919, I was temporarily transferred to the Station Hospital at Ahmednagar. Two days later I was put in charge of the sick parade of the East Yorks stationed on east ridge at Ahmednagar, and, on my first inspection, I saw twelve cases of the same disease as that noted, and each day following I saw from six to eighteen similar fresh cases.

Onset.—Usually sudden, with severe frontal headache, giddiness, or actual fainting while on parade.

Symptoms.—Temperature varied between 99° and 100° F, in several cases reaching 103° F. Pulse about 96, but varied as temperature.

Physical Signs.—The same or the following day the rash began to show as a few circular *discrète* spots on the wrist or lower forearm. The next place it was observed was on the back of the neck, just about the line of the shirt or slightly above it. These spots increased in number and spread up the forearm, arm, chest, back, thigh, and legs, till these were covered. The size of the spots varied from a pea to a two anna silver piece in size. These papules were not raised, and in the early stages were of a pink colour and faded on pressure; later, they became a browner tint, with edges more clearly defined, and did not entirely fade on pressure. In about 5 per cent. of these cases a few scattered vesicles appeared, chiefly on the arms; but I never saw more than half a dozen in any single case—and this was negligible as compared with the number of papules. Only in one case did I see a vesicle the size of a shilling (bulla). I punctured the vesicles, but in the laboratory at Ahmednagar the contents were found to be sterile and the fluid corresponded to an ordinary blister. I never saw a vesicle become pustular in any case.

Pruritus.—Rather less than half the cases suffered from intense pruritus, as shown by the way their arms had been scratched; the remainder of the cases made no complaint on this score. The rash persisted, though fading out when I left Ahmednagar on September 11, 1919, and my early return to England prevented further observations.

I have seen no cases of dengue or three-day fevers with any clinical resemblance to the above. Venereal disease may be excluded, and, owing to complete absence of vesicles on the face and the low percentage with vesicles at all, chicken and small-pox may be put out of court. In conclusion, I would again call attention to the fact that all the cases I saw were limited to the East Yorks regiment, and this regiment had newly arrived in India. I failed to get any history of rash or infectious disease during the voyage out.

Through THE PRACTITIONER I should be glad to hear of anyone who may have seen similar cases, for, up to date, I have seen no account corresponding to the above disease.

A Method of draining the Stomach when the Pylorus is not obstructed.

By DONALD M. MACLEOD, L.R.C.P., L.R.C.S.
Acting Hon. Surgeon, Evesham Hospital, etc.

IN cases of gastric ulcer when the pyloric opening is normal and posterior gastro-enterostomy has been decided upon, a very good method of drainage of the stomach is, not to make the incisions into the stomach and bowel of equal lengths, but to make that in the jejunum longer than that in the stomach.

One of the main causes of the ultimate return of the old symptoms some time after gastroenterostomy we know to be due to contraction and stenosis of the artificial opening, allowing thereby little or no drainage of the stomach contents.

To prevent this as far as possible, I always make the opening in the jejunum at least half an inch longer than that in the stomach. By this means, no matter what the ultimate size of the communicating opening is, there is always a tendency for this opening to remain and be kept patent.

In the usual operation in which the incisions are equal in length, the contractions passing over the stomach tend to close this opening, and so at the same time constrict that in the jejunum. But the complete closure of the communication is prevented by the fact that the contractions have an additional force to overcome, viz., the jejunum orifice. There is, therefore, some degree of patency but not enough, as it were, to invite the chyme into it. For this reason, only a portion of the contents will pass through the new opening, while the rest passes onwards through the pylorus.

The stomach, being in a constant state of "tone," may be compared with a rubber tube and, placed next to another tube, the whole may almost be likened to the relative positions of stomach and jejunum when the operation is performed. Incisions of equal lengths in these two tubes, because of that very equality, remain almost closed. Material of the consistency of chyme, passing slowly along one tube (stomach) without obstruction, will only partly escape through the opening into the other tube (jejunum), whilst the rest passes onwards. This is what actually happens in some cases sooner or later.

When, however, the opening in the jejunum is longer than that in the stomach, contractions which would otherwise close the opening, in the stomach, are prevented from doing so by reason of the fact that, attached to the stomach opening, is a greater bulk, as it were, of jejunum, and this in itself will prevent closure of the anastomotic opening, while this larger mass will at all times keep the artificial opening patent and ready to receive the stomach contents as they pass along. The puckering of the jejunum produced by this method acts as an additional factor in maintaining the patency.

The difference in drainage between the two methods can be well demonstrated by means of bismuth meals and X-rays.

This method need only apply to cases in which the pylorus is not obstructed in any way. Where obstruction of any kind does exist, the usual incisions, provided they are large enough, suffice.

TECHNIQUE.

After the usual preliminaries, a good grasp (at least 3½ to 4 in. of a suitable site) of the posterior wall of stomach, as low down as possible, is obtained and the clamp applied.

Having made the usual observations as to the direction in which the jejunum runs, the highest part is grasped by a clamp which encloses a segment of bowel 3½ to 4 in. long—the clamp enclosing mesentery as well. (The clamp, though useful, is not absolutely necessary.)

The posterior serous suture is begun, and the two parts approximated for at least 3½ in. Then an incision 3 in. long into the stomach is made, and also one into the jejunum 3½ in. long, *i.e.*, ¼ in. longer at each end than that in the stomach. After removal of some redundant mucous membrane, the posterior cut edges of stomach and jejunum are brought together with a single silk suture at each end, and also one joining the middle point of each edge. These three sutures are simply inserted to be a guide to the spacing of the stitches, in order to bring the unequal edges more accurately into apposition.

This having been done, the ordinary posterior continuous suture is introduced, uniting the two cut edges, but the stitches in the jejunum should be spaced farther from each other than those in the stomach. Although some bulging of the mucous membrane of jejunum is to be seen here and there, this can easily be dealt with by inserting one or two stitches.

After this, the anterior cut edges are dealt with and united in the usual manner, after having inserted one stitch in the middle of the two edges.

The anterior serous suture is then proceeded with and the operation completed.

Practical Notes.

The Therapeutic Value of Injections of Milk.

In a recent thesis, Para brings forward a new method of treatment, the subcutaneous injection of milk. The first to use such injections were some ophthalmologists in Vienna, in 1916, for the treatment of iritis and keratitis. In France, too, the method was first used in eye work. Domec, of Dijon, obtained striking results in severe traumatic ulcers of the cornea, in infectious irido-choroiditis, in dacryo-cystitis, and in parenchymatous keratitis of hereditary syphilis. Not only was a cure obtained in cases in which the sight appeared to be hopelessly damaged, but the improvement, especially the disappearance of pain, was obtained almost at once.

The use of the treatment was extended to other infections, especially to the influenza epidemic of 1918. Cordier, and later Gallon, had very encouraging results at Dijon in several patients. Para treated, in November and December, the influenza cases occurring in the division in occupation of the Grand Duchy of Luxemburg, and all his cases of very severe influenzal broncho-pneumonia recovered under this treatment.

The method simply consists in giving subcutaneous injections of boiled milk, 5 cc. for an adult and 2 cc. for a child. The injection is followed with shivering and a slight rise of temperature, but a reaction is only severe in those affected with pulmonary tuberculosis, and this is the only contra-indication for its use.

(*Journ. de Méd. et de Chir. prat.*, November 25, 1919.)

A "Fourth Symptom" for Hutchinson Triad.

Cantonnet insists upon the value of a new symptom to be added to Hutchinson's triad. This consisted of interstitial keratitis, the special deformity of the teeth, and deafness, but arthropathies were hardly mentioned. During the last few years it has been shown that these last named, especially in the form of hydrarthrosis, are very frequent. The hydrarthrosis occurs in the large joints, the knee-joint in particular. It is painless, and will not be observed unless it is searched for. It disappears under mercurial treatment. Cantonnet suggests that the much greater frequency of this symptom should promote it to the triad in place of deafness, which is much less often present.

(*Journ. des Praticiens.*)

Reviews of Books.

On Gunshot Injuries to the Blood-Vessels. Founded on experience gained in France, 1914-16. By Sir GEORGE MAKINS, K.C.M.G., C.B. Bristol: J. Wright and Sons, Ltd.

No branch of War Surgery is more important than that dealing with injuries to the heart and blood-vessels, a subject ably discussed in the volume before us.

The introduction summarizes the experience gained during the Great War, together with that recorded of recent campaigns, especially with reference to injuries caused by modern instruments of warfare. The succeeding chapters deal with the relative frequency of injuries to various arteries and veins, the signs and symptoms together with special reference to the immediate and remote effects of occlusion of the main blood-vessels on the vitality of parts supplied by them. Chapter VIII., on the Vessels of the Neck, includes three tables in which are given descriptions of injuries, sympathetic and cerebral disturbances, prognosis and complications. This chapter will interest the physician and surgeon alike.

As a systematic record and experience of war surgery of the vascular system, Sir George Makins's work is not only valuable at the moment, but it will remain a standard book of reference on the subject.

The publishers are to be congratulated on the get-up of the book and clearness of type. There are 100 illustrations, including four plates, and the index is good.

Internal Medicine. A work for the practising physician on diagnosis and treatment, with a complete desk Index. By JAMES C. WILSON, M.D., and NATHANIEL B. POTTER, M.D. In three volumes. Illustrated with 427 text illustrations and 14 in colour. With Index. Pp. 166, in separate volume. London: The J. B. Lippincott Co. £3 15s. net, the 4 vols.

THIS work forms a complete system of medicine, with the exception that there is little or no mention made of pathology. We would, therefore, suggest that in a future edition a volume should be devoted to this subject, and then the practitioner would have all that is needed for his guidance in Medicine.

The volume on Internal Medicine gives an admirable account of the most approved and modern methods of treatment. A useful feature of the book is the series of tables of mineral waters and their indications and list of drugs. We should also like to draw attention to the numerous prescriptions distributed throughout the volume, which will be of great assistance in practice.

The two volumes on Medical Diagnosis are by Dr. James C. Wilson, assisted by Dr. Creighton H. Turner. Professor Robinson has contributed a section on Graphic Methods in the Study of Diseases of the Heart; Professor Sweet has revised and enlarged his section on Diseases of the Eye; Dr. Lloyd has brought the section on Diseases of the Nervous System up to date; Dr. Rehfuss has devoted attention to the newer Gastro-Enterological Methods, and Dr. Corson White has written an able and practical article on Serology. These volumes are the fifth edition of Dr. James C. Wilson's work on Diagnosis.

Though a very good account of Erysipelas is given in Vol. II. of Diagnosis, no mention is made of it in the volume on Treatment. This is certainly an omission which should be amended in the next edition.

Having the index to these volumes in a separate book is a very convenient arrangement and, after carefully testing it, we find it quite reliable.

We can hardly emphasize sufficiently how much we are pleased with the work altogether; we very highly recommend it to practitioners and students of medicine, and further we cannot conclude the review without expressing our admiration on the way the publishers have fulfilled their task. The paper is good, the printing very clear and distinct, and the illustrations are of a very high standard of excellence, the coloured illustrations being works of art.

Studies in Electro-Pathology. By A. White Robertson, Temp. Major, R.A.M.C. Pp. x + 304. London: Routledge and Sons. 12s. 6d. net.

After some introductory remarks on the "law of the wild," the author passes on to consider the nature of light, some factors in metabolism, and electric phenomena in cells and in metabolism. Various interesting suggestions are made in these sections, *e.g.*, that the function of phosphatides is to energize oxygen, that the antiseptic action of certain aniline dyes is due to their power of absorbing chemical energy from the blue-violet rays, and that they may thereby oxygenate the tissues. Dielectrics (insulators) and dielectric tissues are next discussed. According to Baines (upon whose work much of the matter in this volume is based), the tissues possess an electrical potential, which is normally maintained more or less constant by generation of charge through nerve force and dissipation of charge to air. Wound-treatment is next considered. It is stated that the area of the wound at an early stage is an area of diminished electrical discharge, but when reaction is established electrical discharge is increased much above the normal. The author, therefore, considers that the bacterial infection may be neglected, and that the aim should be to prevent loss of current. This he does by the use of paraffin or of rock oil dressings—dielectrics. The efficiency of "Bipp" paste he maintains is chiefly due to the dielectric—the liquid paraffin—present. A number of cases treated successfully with rock-oil dressings is recorded. Other diseases—beri-beri, tuberculosis, pneumonia—are subsequently considered from the same standpoint. Sufficient has been said to indicate that the volume contains matter of very considerable interest, somewhat hypothetical, it is true, in many places, but not, therefore, to be summarily dismissed.

Studies in Electro-Physiology (Animal and Vegetable). By ARTHUR E. BAINES. Pp. xxix + 291. London: Routledge and Sons.

THE author of this book is a submarine-cable engineer, and years ago, when testing cable, observed a galvanometric deflection for which he could not account; upon investigation, he found the disturbing influence to proceed from his own body. From this incident a number of experiments were initiated upon the electrical properties of animal and plant tissues, and the present volume is the outcome of this work. The author finds that everything living, whether animal or vegetable, has a well-defined electrical system. In fruits and vegetables the edible plant is the positive element, the skin or peel being of the nature of an insulating substance. As a matter of fact this is no new discovery, and the botanists are well aware that a difference of electrical potential exists between the different organs, parts of organs, and even parts of the same cell, in a plant.

Numbers of experiments demonstrating the existence of these electrical phenomena in animal and plant tissues are recorded. From these a universal "law" is deduced, but what this law may be is difficult to ascertain—it seems nowhere to be clearly formulated. Apparently it is that the living organism is electrically a "condenser" and all its parts—organs, tissues, cells, etc., are condensers. The whole book and argument is very involved and difficult to follow. The book is well produced, and illustrated with several coloured plates and many text figures.

Vicious Circles in Disease. By JAMIESON B. HURRY, M.A., M.D. Third and enlarged edition. Pp. xx + 377. 22 plates. London: J. and A. Churchill. 15s. net.

DR. HURRY is no stranger to readers of THE PRACTITIONER, for many of his articles dealing with this subject have appeared in our pages. We are sure that our readers will welcome the opportunity of further cultivating their acquaintance with a subject which the author has invested with so much that is of interest, as well as of practical help and of importance in the daily work of our profession, both in diagnosis and in treatment. The subject is dealt with systematically, and, by so doing, Dr. Hurry opens up new avenues of thought on the processes of disease which lead to points of view revealing positions of greater advantage from which to attack the problems involved. The numerous references to current literature and to leading authorities afford evidence of the great care and incalculable labour that have been devoted by the author to the preparation of his work.

Preparations, Inventions, Etc.

DIGALEN GRANULES.

(London: The Hoffman-La Roche Chemical Works, Ltd., 7 and 8, Idol Lane, E.C. 3.)

In this preparation each granule contains $\frac{1}{1000}$ gr. of digitoxin (Cloetta). Digalen is a standardized solution of amorphous digitoxin, and represents the full activity of the digitalis leaves, which cannot be claimed with certainty for the galenical preparations of the drug. It is comparatively quick in action, and is eliminated fairly soon, so that non-cumulative effects do not occur when used for treatment continued for some time. The small dose contained in each granule allows the individual dose suitable for each case to be determined easily and definitely.

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This diary is very convenient and useful. It is handy, compact, well suited for the busy medical man's table, and the price (2s. 6d.) is low. We can recommend the diary to our readers, but we should have preferred less advertising matter throughout.

THE PRACTITIONER

FEBRUARY

1920

The Phantom Limbs of Amputés.

By EDRED M. CORNER, M.C., F.R.C.S.

Surgeon to St. Thomas's Hospital, Consulting Surgeon to Queen Mary's Auxiliary Hospital, Roehampton, etc.

IT is an old observation that people who have undergone an operation of amputation have sensations in the parts that have been removed. In spite of the age of the observation, little has been written on the subject since the elaborate contributions of Prof. Pitrés and, later, of Luys. In consequence, and with the aid of Sir John Lynn Thomas, to whose kindness and energy I cannot sufficiently acknowledge myself indebted, an elaborate survey of these surgical phantoms was conducted. In all, particularly with the aid of Dr. Calvo, Mr. Liang, Mr. Curwen, and Mr. Tattersall, over 500 cases were investigated. These enquiries established these facts:—

1. Phantoms made their appearance immediately after operation.

2. They were very unusual in the young, but were more frequent among the older.

They are of much greater frequency and severity in military than in civil practice.

3. As a rule, they obtruded themselves less in frequency and sensation as time went on, and within eight months or a year the patient slept well, did not dream, and was only conscious of the phantom limb when he thought of it. It was a sign of con-

siderable clinical importance if the patient was undisturbed by his missing limb in sleep and unaffected in dreams.

4. There was great confusion in the answers as to the limb in dreams. In ordinary life the man may have lost one limb, yet in his dreams he had both. Others varied still more, sometimes having one and sometimes both. Such conditions strongly suggest that there can be no local lesion of nerves. In some dreams the man has even lost the wrong limb! These conditions must arise from the mind; they are central. Others seem different. Some always have two limbs, but the one which had been amputated seemed awkward; one dream always ended in the man hurting himself and awakening. Another, who had in his dreams the full complement of limbs, felt pain at the site of the amputation. Such cases suggest a local lesion which directed attention to the amputation.

5. The character of the sensation was usually cramplike or crushing. Occasionally it was scalding. But these sensations, like those of electric shocks, were momentary or of short duration.

6. As a rule, the phantom limb could be neither bent nor moved. Thus, the phantom was an abnormal limb. One man found his phantom leg short, and it used to swing beside him as he walked.

In their later stages, these patients become mindful in their unconscious state that they have only one limb, and dream accordingly. Thus, the early "bipedal" dreams become "monopedal."

7. Usually, in time all sensations of a phantom limb disappeared; but, when it persisted, the man grew very depressed and even homicidal. From time to time in such a man, and after wearing an artificial limb, the sensations and the dreams return. A badly-fitting. unsuitable limb has possibly irritated the

newly-grown nerves in the stump.

8. Operation frequently failed to cure the sensations in a phantom limb, suggesting the central origin of these sensations. More frequently operation, if on the right place, altered the sensations but did not abolish them, temporarily increasing them, from the traumatic stimulation of the nerves at the operation. For example, before operation some had the power to move the phantom limb; operation made the limb immovable and unbendable.

9. The factors which most frequently affected the sensations, were cold, damp, changes of weather, and absence of occupation for the mind (such as results from prolonged stay in hospital).

Men, undoubtedly, brood over the loss of a limb, and perhaps such patients are more prone to suffer from mental depression than others. At any rate, neurasthenic complaints are very frequent amongst them. Hospital life is bad for such patients.

10. The distribution of the sensations are often not in the anatomical area of a nerve or nerves, and by their variations suggest their central origin.

11. The nearer the trunk, particularly in shoulder amputations, the more persistent these sensations are apt to be. The shoulder and high arm amputations seem to be especially troublesome.

12. A heavy, ill-fitting limb, by its irritation, can bring back phantom sensations. The mere wearing of an artificial limb may do this.

It would appear that these sensations, being easily observed, are worthy of clinical study. There has been an idea for a long time that the sensations along a nerve were "projected" into the phantom, and there seems to be some truth in this. The phantom is itself of central origin; whether the sensations arise in cortical or subcortical changes, as yet we cannot say. It is fully known that the removal of a limb is followed in the central nervous system by changes of degeneration right up to the cortex. Sir John Lynn Thomas told the author of a case in which a laminectomy and division of the spinal roots failed to relieve the pain, the source of which was obviously higher. Conversely, an officer (T.), at the 5th London General Hospital, had a stump too painful for fitting. Massage and movement completely cured his pain and phantom in three weeks. There is no need to comment on the central origin of both his pains and his phantom. A careful study of such a case sometimes reveals the fact that there is not a constant sensation in the distribution of a nerve, suggesting that there is a source of irritation on that nerve giving rise to the sensation which is projected into the phantom. Thus, some cases can be relieved and cured by surgery; but, in general, all that can be hoped is that operation will alter the phantom sensation and, in the course of time, lead to its cure.

Some phantom sensations or pains may persist for years and, affecting life and health but little, do not demand special treatment. The brain normally receives impulses from a limb, thereby gaining worldly experience; and, being deprived of these by the removal of the limb, the organism may take long to accommodate itself to the new conditions. The cortical and subcortical degenerations and, perhaps, regenerations, may themselves give rise to sensations interpreted as pains. Who knows? The following case suggests the possibility that repair foci may give rise to sensations.

A corporal had his leg shattered in September, 1916. His leg was removed by a guillotine amputation the same day. It was re-amputated in October, 1916. Distressing pains followed, and the nerves were removed in April, 1917, and the scar excised in August, 1917. Pains persisted and a sciatic neuroma was removed full of foci of repair rather than of foci of acute infective inflammation. The neuroma was adherent to the hamstring muscles, but had not invaded them. This was in November, 1917. The pains were better but persisted, and in January, 1918 he had a chain of these regeneration neuromata removed with a bulb on the lower end of his internal saphenous nerve. After this he was better, but not well. The stump still showed constant tenderness along his femoral artery. In April, 1918 the contents of Hunter's canal (femoral artery and femoral vein with their silk ligature and internal saphenous nerve) were removed. There was found a focus of repair at the end of his internal saphenous nerve. Its appearance and situation suggested that it might have arisen from the irritation of a silk ligature on the artery. Another regeneration neuroma was removed and examined. It showed that it consisted largely of fibrous tissue and without a focus of irritation within it. It was harmless as a source of trouble. Later the scar was excised, and all local pain vanished. The stump could be freely handled without causing pain. Only the mental, or central, factor remained. The man was treated by hypnotic suggestion, sent back to work, and ultimately got quite well.

These pains may persist and the patient be unable to get away from them, getting steadily worse and ultimately, perhaps, becoming dangerously insane. The treatment of these cases is most difficult, and in deciding treatment two points need constantly to be kept in mind:—

> Only operate if there are constant unvarying local physical signs.
>
> The complaints of the great majority are central *in toto* or *in parte.*

To show how very difficult it may be to decide about a patient, two letters, each from a nameless and great authority, about the same patient with recurrent pain in his shoulder, and who ultimately got quite well, are given:—

I.—" I should think that, as the patient says, the pain is growing worse, fresh regenerating fibres are growing down, and I observe that there are continuous contractures of the muscles above the stump which are, no doubt, full of these regenerating fibres, and this gives rise to the pain which he projects into his hand. Both the median and the ulnar distributions are equally affected, consequently, if it is proposed to do the Förster operation of division of posterior roots, fifth, sixth, seventh, and eighth cervical and the first dorsal would have to be divided.

This is a very serious operation and should not be undertaken unless the pain becomes unbearable."

This entirely different opinion was given on the same patient:—

> II.—"He now complains of shakiness of the stump, discomfort in the end of the stump, and feelings of the right hand, which seems to be clawed and drawn up to varying amounts. The pain in his hand seems to affect the thumb to a greater extent than the fingers. At times he feels the wounds near his elbow, and at others a sensation of cold water running down the arm bothers him. At night time he sleeps badly, and then his right hand is more troublesome than at other times.
>
> Since his discharge from the Army, he has lost a considerable amount of interest in things at large; he has played few games, and his office-work does not appeal to him as of yore. On examination, over-action and shakiness are constantly present in the affected stump. He finds the muscles of the right shoulder girdle difficult to relax; there are no tender nerves.
>
> I am of the opinion that psychological factors in this case are more important than physiological or anatomical. I should like to suggest this man undergo a training, of *both* upper extremities and be made to do a series of exercises à la Sandow, and should be encouraged, despite all complaints on his part, to wear his artificial limb. He is an intelligent fellow, and, if he trains himself, ought to be able to make a considerable amount of use of an artificial limb. I have talked with him in this strain, and hope that he has been impressed thereby."

In the end, without special treatment, the man got quite well; the operation allowed him to wear his artificial arm and the man himself, returning to work, conquered the central sensations. It is wrong, wickedly wrong, to herd these people together in hospitals. Let them be got to work.

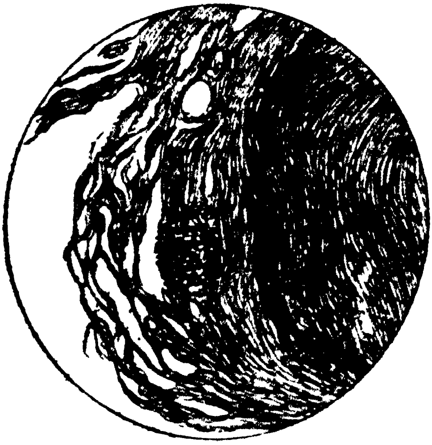
Professor Marinesco, of Bucharest, showed that when the nerve fibres regenerate, they branch, divide, join up with other bundles and behave in many ways, such as by re-inervating and invading structures, like a malignant growth, complicating enormously the sensations passing through them for the brain to interpret. This will explain the great difficulty in diagnosing and treating successfully these patients, as illustrated by the two preceding letters. Moreover, it is easy to understand how the tales of pains vary, and, especially in a partially educated man, may contradict each other. The ways of regeneration of nerves must make all listen sympathetically to these tales, realizing that there is "a grain of truth amongst the chaff," and only trying to see that grain.

To the clinician, there can hardly be a stronger argument, that foci of repair can cause sensations projected by the patient's mind into the phantom, than the common history of troubles and pain in the lost limb getting less and less as time goes by and the changes of repair become less and less active. Such patients dream less and less; in their unconscious state, the amputated limb asserts itself less and less. The phantom limb seems to be central in its origin, but the variations in its sensations may be peripheral. It is only when there are very decided peripheral signs, not symptoms, that operation is likely to be of use.

At the commencement, immediately after the operation, all suffer from some sensations such as are due to dividing the nerves at the operation, and in their dreams possess the amputated limb. Later, the pains of the lost limb decline in severity, and still later consciousness of the lost limb takes no effect on the dreams, the sufferer now possessing only one limb even when unconscious.

I should like to mention one particular phantom, for it may be of clinical value. Some patients complain that they cannot feel their toes, but their insteps feel as though crushed in a vice. At times they forget this sensation and sleep well. At other times, the sensation persists, and is not affected by operation on the great sciatic nerve. But, apparently, it is affected by removing the femoral artery from Hunter's canal with scar tissue or silk ligature on it and the internal saphenous nerve adherent to it. Therefore, the presence of a tarsus-crushing phantom, an adductor spur and tenderness along the femoral artery is an indication that operation should be considered.

The presence of or variations in the phantoms are often of considerable clinical value, particularly in conjunction with other physical signs, in distinguishing whether the symptoms in a particular patient are physical or psychical, bodily or mental, peripheral or central.



Inflammatory nodule in the end of the bulb of the internal saphenous nerve removed from Hunter's canal in a patient in the King George Hospital with a very troublesome painful stump. Ultimately the man got quite well.

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Painless Operations.

BY P. LOCKHART-MUMMERY, F.R.C.S.

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IN the early days of surgery before the discovery of anæsthetics, an operation of any kind was always accompanied by dreadful pain, and in these days it is difficult to realize what the unfortunate patient, who had to undergo an operation, must then have been called upon to endure. No one of this generation can remember those days, and we can only surmise what occurred by the descriptions given by the surgeons and, sometimes, by the historian of those times. We have not very many accurate descriptions of operations from the patient's point of view in the days before anæsthetics, but we can be in no doubt about the pain which a patient must have been called upon to put up with. Only the strongest survived, and nothing but sheer necessity made the patient submit himself to the surgeon's knife. Amputation was performed in the Napoleonic wars on the field with nothing more than the necessary instruments and a few simple dressings. The surgeons of those days developed a dexterity and speed that we do not attempt to emulate, for the necessity for great speed, fortunately, no longer exists.

The discovery of anæsthetics did much to do away with the worst horrors of an operation, and the minds of medical men then turned themselves to the task of enabling patients, who had been operated upon, to recover safely and with the minimum risk. Lister's great discoveries were the next big advance, and when the present generation of surgeons was first introduced to the operating theatre, operations were of common occurrence, and it was quite the exception for patients to fail of recovery, or to have inflammation in their wounds.

While anæsthetics have entirely removed pain during operations, and antiseptic surgery has, by preventing inflammation, done much to prevent pain during the process of healing, operations are still, in the public mind, associated with pain and discomfort, and not entirely without reason. My endeavour will be to interest the reader in the question of preventing *all* pain from an operation; under this heading I will also include discomfort, and point out the ways in which this can be done.

It is necessary, first of all, to refer briefly to the nature and causation of pain, for no one, who does not clearly understand what I may call the physiology of pain, can succeed in preventing it. In spite of what one may think to the contrary, pain is a beneficial thing, entirely in the interest of the individual, and developed during the course of ages for man's protection. The more highly developed the race, the more sensitive to pain will its individual members be. The absence of pain would be a fatal calamity, and one can easily realize this from a few examples. Paralysis of the corneal branch of the fifth nerve results in loss of the corresponding eye. The fact that the entrance of a small particle of dust or other irritating substance into the eye fails to cause pain, and so induce means for its immediate removal, results in the particle setting up ulceration of the delicate conjunctiva and in consequent destruction of its substance. As is well known, loss of sensation in the foot or hand results in ulceration of the fingers and toes. One of the best examples, perhaps, is in relation to the X-rays. As is well known, X-rays cause burning just as do heat rays. But the X-rays are quite a modern discovery, and man has not had time (millions of years being necessary) to acquire any protective sensation against them, with the result that he gets burnt without being aware of the fact until the ulceration appears. Imagine how dangerous heat would be, if it failed to cause pain when in excess.

Pain, then, is protective, and since Nature wastes nothing we are only provided with sensory nerve endings in those tissues without protection from outside injury. The wall of the intestine contains no sensory nerve endings, and we can crash or burn the bowel wall without causing any sensation. Obviously, surgeons were not provided for in the course of evolution, and an injury which damaged the intestine would be fatal, so it would have been a waste of time to provide nerves in the bowel wall. Sensory nerves are, however, present in the mesentery, but they are not tactile. Stimulation of these nerves produces what is known as "stomach-ache." The object of these nerves is to prevent over-distension of the intestine, and should over-distension take place they immediately cause the characteristic pain. Cutting the edge of the mesentery during an operation under local anæsthesia causes stomach-ache, not ordinary tactile pain.

The deeper tissues beneath the skin are not at all freely supplied with nerve endings as compared with the skin. But we still have much to learn about the exact anatomy of nerve endings in the body. The distribution of the sensory nerves in muscles and in the fascial planes is little understood. More is known about the peritoneum, though our knowledge is very imperfect. We know that the parietal peritoneum is highly sensitive to injury, but that the visceral peritoneum is mostly insensitive.

To prevent pain or discomfort after an operation is a complicated business, and can only be achieved with a good deal of trouble and patience; but it can be done. Much depends upon how the operation is performed. Tissues should be separated only by clean cutting with a sharp knife, and crushing or tearing must be entirely avoided. The wound must not be stretched open with retractors; large pieces of tissue must not be caught up in artery forceps; when it is necessary to clip a vessel, the vessel only should be caught if possible; blunt dissection should not be done; everything must be handled lightly, including swabs. I always regard the wound as if it was made of the thinnest and most brittle glass, of which it is a crime to break any part. The wound should not be washed out, even with water, and the stitches should be as few as possible, and should not be tied tighter than is necessary just to approximate the edges. Vessels should be twisted and not ligatured. One must always bear in mind that the least roughness will be paid for by the patient in pain after the anæsthetic has passed off. Unless one can operate on these lines, other precautions will be useless or will only partly achieve their object.

The anæsthetic is extremely important. If the patient is going to vomit after the operation, he is going to have much distress, for there are few things so distressing as being sick, and apart from that the effort of straining tends to tear the wound and make the stitches cut in. Post-operative vomiting must be done away with if we are to realize our ideal. This can be achieved by using local anæsthesia only, or spinal or regional anæsthesia, either with or without a general anæsthetic such as gas and oxygen, or in combination with scopolamine and morphia anæsthesia —"Twilight Sleep." By a careful combination of these, one can generally succeed in obtaining an almost perfect anæsthesia which will not cause sickness.

The important point about the general anæsthetic is that instead of the gas or drug being used, as hitherto, to produce anæsthesia, it is only given in sufficient quantity to produce unconsciousness (analgesia), sometimes hardly that. The ideal condition is the one in which the patient will react to stimuli, will answer if spoken to loudly, or move if hurt. I always ask the anæsthetist to let me know if he sees any sign of stimulation, for this is evidence that the local and regional anæsthesia is not efficient.

There are several drugs which can be used to obtain local anæsthesia for varying periods, but the ideal drug has not yet been found. I generally use a 1 or 2 per cent. solution of novocaine with adrenalin. This solution is freshly prepared for each patient in quantities of 50 cc. It is sterilized twice over to make quite sure that it is sterile. As much as 50 cc. may be employed at a time. A special type of syringe is used for injecting it, and Sheil needles of various sizes.

A new drug which I am now trying is "apothesine," the correct name for which is "gamma-diethyl-amino-propyl cinnamate." It is not a cocaine derivative, and it is said to be more than twice as effective as novocaine, and less toxic. Cocaine itself is not safe unless used in very weak solutions, 0·5 per cent. and under, and with adrenalin, the object of the adrenalin being to prevent rapid absorption of the drug by constricting the vessels in the neighbourhood of the injection. Eucaine B may also be used, but seems less effective than novocaine. The anæsthesia from these substances is produced in about 5 minutes and lasts from 45 minutes to an hour, according to the amount injected.

Quinine urea hydrochloride in 0·5 per cent. solution can be used to prevent after-pain when from the nature of the operation this is to be expected. It should be injected with long needles into the tissues around and beneath the wound. So far as possible it should not be injected into the immediate neighbourhood of the wound, for it is rather irritating and may produce a temporary œdema, and delay healing. It produces anæsthesia in from 20 minutes to half an hour, but the anæsthesia lasts about two days. When carefully injected the results are quite surprising. I remember one patient of mine who had his appendix removed, and in whom I used this substance, who, 24 hours after the operation, could blow his nose violently without the slightest feeling of discomfort in the wound.

Anyone who wishes to become expert in the use of local anæsthesia for ordinary operations will be well advised to begin by doing operations under local anæsthesia only. It is only by so doing that one can learn the method and find out just what is required. If one begins by using gas and oxygen, or "twilight sleep," in addition, the bad results may be attributed to the latter rather than to the inexpertness of the surgeon. It is possible to perform almost any operation under local anæsthesia, and during the war Colonel Grey of Aberdeen performed most extensive operations at the front with local anæsthesia only. Considerable care and a certain amount of patience are necessary, not to speak of an accurate knowledge of the anatomy of the part. One must leave time for the drugs to act before beginning to cut, and light handling is essential throughout.

There are many other details which require attention; many of them are not very important in themselves, but, added together, make all the difference between success and failure. How often is any attention paid to the comfort of the patient on the operating table? In our desire to have a clean operating table, too often we compel the patient to lie in one position on a very hard steel surface, with nothing but a thin blanket or a mackintosh between him and the metal. It is interesting to note in passing that the mackintosh is not put there to protect the clean patient from a dirty table, but to protect the clean table from the dirty patient, and, incidentally, to save the nurse from having to clean it. A comfortable position on a well padded table is necessary, or the patient will have vague pains in his legs and back afterwards, for which it is now usual to blame the anæsthetist. I do not quite know why, but probably because he is not there to defend himself.

The improvement in results which follows what I may call "painless operating" is quite surprising. Several things follow from it. Since the patient's digestion is in no way upset and he is not going to be made sick, there is not the slightest occasion to purge him, or starve him, either before or after the operation. My patients who have had an abdominal operation do not, as a rule, miss a single meal, nor is their diet modified; they are given the ordinary food to which they are accustomed throughout. Healing is more rapid because the supply of the necessary material is in no way interfered with. Secondary hæmorrhage is much less likely to occur, since there is no straining from vomiting to excite it. Other complications are equally absent for the same reason. The benefit that results is particularly noticeable in old people. Children and elderly people stand starvation very badly, and it is particularly advisable to avoid any alteration in their diet. The digestion of elderly people is easily upset by ether or chloroform, and bronchial trouble is also easily excited. In the absence of inhalation anæsthesia elderly people seem to stand operations quite well, but it is most important that they should never be allowed to get ill.

This method of operating has made it possible to operate on old people with an impunity that one had not supposed possible. I have performed quite serious operations on patients as old as 75 without their causing the least anxiety. Most surgeons now make use of similar methods when operating upon old gentlemen with enlarged prostates.

Spinal anæsthesia is very satisfactory in operating upon elderly persons, but I consider it of the utmost importance that the patient should be carefully handled while under the influence of the drug. Spinal anæsthesia puts out of action the vasomotor control to the lower part of the body, and unless the patient is kept throughout the entire period of anæsthesia in a position with the head lower than the feet, complications are liable to result. I always see that the patient is kept really head down till the anæsthesia has quite passed off. If this precaution is taken, no trouble from collapse or headache need be anticipated.

Another important point to remember when operating upon elderly people is not to confine them to one position. It is advisable to encourage them to sit up and to move about, even to get out of bed after the operation so that their circulation is kept going. Daily light massage after operation is often useful, and is not used as frequently as it should be.

I should like to observe, in conclusion, that much of the discomfort from which patients used to suffer after an operation was due to sheer folly. When I was a house-surgeon it was the practice for patients who had had an abdominal operation to be placed in bed with no pillow, with sand-bags on each side of them, and with their knees tied together. They were not allowed to move from this position for two or three weeks. For the first 48 hours they were given nothing by the mouth but sips of hot water, and after that only a slop diet of beef-tea and milk. It is now obvious to us all that there is no sense in this. Movement can do no harm, and it is essential to comfort. The position of greatest comfort is also that of greatest rest, and this is what is wanted.

The same principle applies to the question of diet. We do not want the patient's digestion upset. Then, why put him on a slop diet of milk and beef-tea to which he is quite unaccustomed, when his ordinary diet is not causing indigestion? In other words, why try experiments with a patient who has just undergone an operation? When the operation involves the alimentary canal many surgeons still seem to think it necessary to insist upon a liquid diet. One can only assume the argument is that if the diet is liquid when it passes the teeth it must also be liquid when it reaches the anastomossis in the alimentary canal. A totally fallacious argument, as we know! Milk which is liquid when swallowed is probably the least liquid kind of material when it has been five minutes in the stomach. All ordinary food is quite liquid when it leaves the stomach and enters the small intestine, and by the time it reaches the cæcum there is no difference as regards the solidity of the residue between a beef-steak and a bowl of soup. There is, however, a good deal of difference as regards the amount of nourishment the patient has obtained, and it is in favour of the beef-steak.

Medico-Legal Notes.

(continued)

By SIR JOHN COLLIE, C.M.G., M.D., J.P.

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THE Courts have decided that when a workman is being medically examined for the purpose of the Workmen's Compensation Acts, he is not entitled to have present at the examination either his solicitor or his friends. Advantage should be taken of this ruling to exclude from the room everyone except the Claimant's doctor. In no case should the solicitor conducting the case, or the paid agent of a Trade Union, be allowed to be present. The reason for this is obvious. In the event of it becoming necessary to report that the symptoms complained of do not exist, or that the case is fraudulent, an attempt would be made to discount the report by making allegations that the examination was incomplete, or even bringing charges of harshness against the examiner. I recall an occasion on which a third party was incautiously allowed to be present during an examination; he turned out to be a clerk in the employ of the claimant's solicitor, and, later, appeared as a witness at the trial, and endeavoured to bolster up the claimant's most unfounded allegations.

It is important, however, to remember that in common law and employers' liability cases no such salutary rule operates.

The first thing to get from the plaintiff at the examination is his account of how the accident happened and what followed.

He should be asked such questions as: Was he able to move without assistance? Did he continue at work for any time? Did he walk, or was he conveyed home, or to a hospital? Was he detained at the hospital? Did he subsequently attend as an out-patient, and if so, for how long? A knowledge of these details may eventually turn out to be very important. At the time of the passing of the Workmen's Compensation Act, *Punch* anticipated the necessity for this class of questions by depicting a motor accident where the motorist, who had knocked a man down, is anxiously inquiring if he was hurt; the answer being: "I don't know until I have consulted my solicitor."

It should be made an invariable rule to decline to listen to any account of the opinion said to have been expressed by any other medical examiner who has previously examined the case, or even by the man's own doctor. It is difficult to see how such information could be useful. I never accept such an opinion unless it is written over a doctor's signature. The accuracy of Dickens's statement that "What the girl said to the soldier is not evidence" has been verified by many judicial references; and what a claimant says a doctor told him is still more remote from being acceptable.

It is often enlightening to obtain a history of the treatment already undergone. An honest workman will generally be found to have endeavoured to obtain the best treatment, and to have followed it conscientiously. The malingerer usually studiously avoids it. If he has been taken to a hospital, he takes his discharge as speedily as possible, and attends the out-patient department (or it may be the surgery of some hard-worked doctor) irregularly, and only sufficiently often to enable him to obtain certificates of disablement.

When writing the report remember to keep clearly before you that the principal thing you are called upon to do is to state *what is the condition at the time of the examination.* Plaintiffs who have been ill, and probably suffered much, but who are well, or nearly so, when they come for medical examination, are usually very anxious to mix up the symptoms which *have been* present with those existing at the time of the examination. The best way to deal with the position is to let the patient clearly understand that you have not examined him before, and that you have only to report on his present condition, but that this does not imply that you do not sympathize with him and appreciate what he must have suffered in the past. I am always suspicious of vague complaints which keep a man indefinitely from work, especially when he will not commit himself even approximately to a time when he thinks he may return to work. Working men sometimes say they will consult their *solicitor* before giving an opinion as to when they may be able to resume work.

One of the most frequent pitfalls for those who are not accustomed to medico-legal matters is dealing with a man who has a genuine, but trifling, injury which has all but disappeared, and who exaggerates what remains so grossly that it almost amounts to malingering. The safe course is to state accurately what you believe to be the extent of the original disability, and to report that at the time of the examination practically all traces had disappeared, but that from lack of will-power the plaintiff is still clinging to the memory of what he had suffered. It is due to the Court to explain that the alleged incapacity is due *now* solely to the memory of what had existed, and not to anything that now prevents a return to work; that, in fact, there is nothing now present which an ordinary healthy-minded person would not ignore. It should be explained, further, that there comes a time in the recovery from all accidents when those who have suffered must, for their own sake, take their courage in both hands, throw off invalidism, and commence work; this alone is the best cure for a condition which is psychical not physical.

The medical examination of women claimants is always peculiarly difficult. They nearly all lose their sense of perspective and proportion, and are sometimes even irresponsible. The instability of their nervous system enables them to stand less strain. Most women resent such examinations, and seem to think that their own statement of what they feel ought to be conclusive, and that to attempt to verify it is an impertinence. I examine few women for medico-legal purposes, which is one of the many things I am thankful for.

The examination of a child who has had an accident presents little difficulty, provided one firmly declines to allow him to be prompted by his parent. Children seldom lie to a doctor, and even if they have been coached they are easily caught out. I well remember the case of a fine little fellow whose parents were claiming £1,400 in respect of a serious injury for which the lad had been trephined. It was alleged he was mentally defective as the result of the operation. I tried him with vulgar fractions; this, happily, was apparently his *forte*. His spelling, also, he was proud of. He distressed his mother by the way he showed off his general knowledge. When I asked him who had won the last two boat races he answered correctly, fairly scoffing at my ignorance. He probably knew the last Derby winner. The mother was very angry—but the claim was settled, I was informed, for £250.

(*To be continued.*)

The Heart in Acute Febrile Diseases.

By H. L. CRONK, M.R.C.S., L.R.C.P.
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ALTHOUGH it is generally believed that in febrile illnesses the myocardium is, to a greater or less extent, weakened, no method of measuring the amount of this weakening has, up to the present, received unanimous approval.

Since influenza is a typical febrile disease of acute onset and short duration, and, moreover, since opinions differ so markedly as to its effect on the myocardium, it was thought that an investigation of cases of this disease might be of some interest. This diversity of opinion is sufficiently shown by the following quotations, from which it is clear that the estimation of the functional power of the heart muscle is very difficult to determine.

Hirschfelder[1] states that, "Just as diphtheria affects the myocardium in the very young, influenza affects it in the aged. Indeed, myocarditis constitutes one of the gravest effects of this disease, and is especially to be feared after the sixth decade."

In THE PRACTITIONER for January, 1919, Sir James Mackenzie[2] says that "The effects of influenza on the heart, in the great majority of cases, is like that of any other febrile affection"; and again,[3] "I would infer that . . . the influenza bacillus does not, as a rule, manufacture a poison detrimental to the heart." In the same article, he quotes one case of sudden death in a patient "not suffering severely" from influenza, and one case of partial heart-block which he is inclined to attribute to vagal action.

In the same issue of THE PRACTITIONER, Samuel West[4] remarks: "It is clear that influenza has a profound effect upon the cardiac muscles and nerves. Indeed, in this respect, influenza has a close resemblance to diphtheria." And again,[5] "Cardiac failure is one of the risks of uncomplicated influenza."

Seeing such contrary views expressed, and reading of Leyton's reliance on the differential stethoscope in diagnosing weakness of the myocardium in patients showing no other definite sign of such a condition, it was thought that some light might be thrown on the hearts of influenzal patients by the same means, and, with this end in view, a number of cases were studied.

Unfortunately, the writer was unable to begin these observations until the epidemic was dying down, so that the cases were all of mild, uncomplicated disease. The results obtained in the 10 cases discussed here are a fair sample of the total (21), and are only selected for the greater number of observations made on them and for the relatively greater fulness of the records.

Previous observations on cases of influenza with the differential stethoscope have been made, but the only references at present to hand are made by Leyton,[6] who states that "The cases of influenza . . . with the ratio of the heart sounds becoming unity, and as recovery occurs returning to 2:1, are so numerous that any record would be tedious"; and by Thorne,[7] who says: "I have found the instrument (differential stethoscope) of great value in judging the effect of acute febrile conditions, such as influenza . . . on the myocardium. The results obtained enable the progress of the disease in the acute stage and during convalescence to be estimated, and the patient to be kept in bed as long as there is any evidence of myocardial weakness." He then gives the following figures for three cases, but does not say at what stage in the disease they were obtained, or whether the cases were severe, mild, or complicated in any way:—

Case 1	-	30 per cent.	50 per cent.	70 per cent.
„ 2	-	0 „	20 „	75 „
„ 3	-	100 „	70 „	100 „

By 100 per cent. he means a ratio of 2 : 1, and by 0 per cent. the writer understands he means a ratio of 1 : 1. He finds that "In many cases of influenza the myocardium is not affected."

In brief, these two observers appear to hold the belief that a lowering of the 2 : 1 ratio is indicative of weakness of the myocardium in an acute attack of influenza, and that although many cases show this lowering of the ratio many others do not.

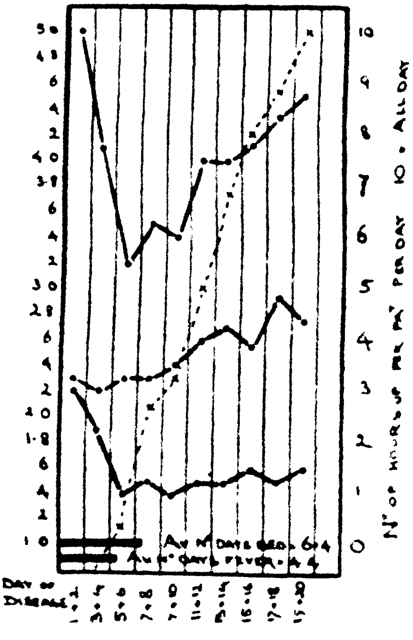

FIG. 1.

THE MYOCARDIAL RATIO IN INFLUENZA.

The results obtained in the present investigation are best seen in the composite diagram (1) made from the 10 cases which were studied intensively.

The readings of the first sound and the second sound with their ratio, which is here called the myocardial ratio, are given for periods of two days combined. On the same diagram are shown a curve of the average number of hours up per patient per day, and also the average number of days' fever per patient and the average number of days in bed. All the measurements were taken with the patient recumbent.

It will be seen from this diagram that the myocardial ratio (M.R.) was high on the first four days of the disease, rapidly fell to a minimum of 1·4 where it remained for six days, and then gradually rose to amaximum of 1·6 with a rapid rise in intensity of the first sound and a smaller rise in that of the second. The high readings of the first four days correspond with the duration of fever, and the rapid rise of the intensity of the first and second sounds with the period during which the average number of hours up per patient corresponded to half the day or longer.

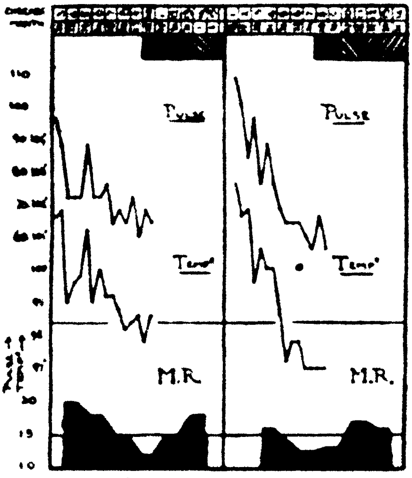

Fig 2.

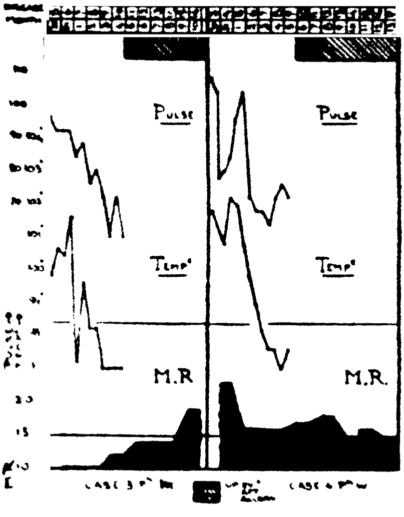

Fig. 3.

As samples of the results from which this composite diagram was constructed, detailed diagrams of the first four cases studied are shown (2 and 3), with curves of temperature, pulse-rate and myocardial ratio, with dates of getting up. The smaller diagrams (4 to 7)

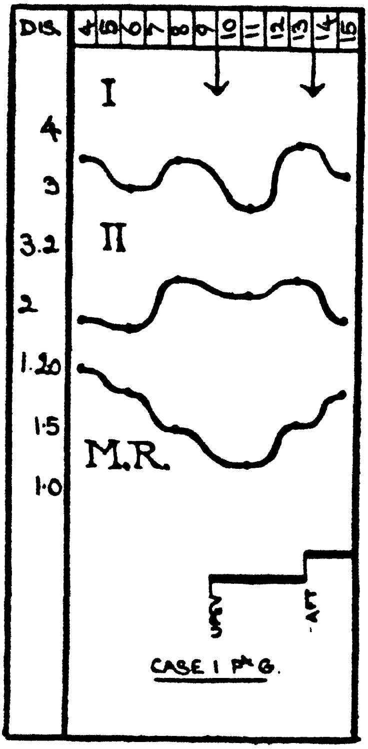

FIG. 4.

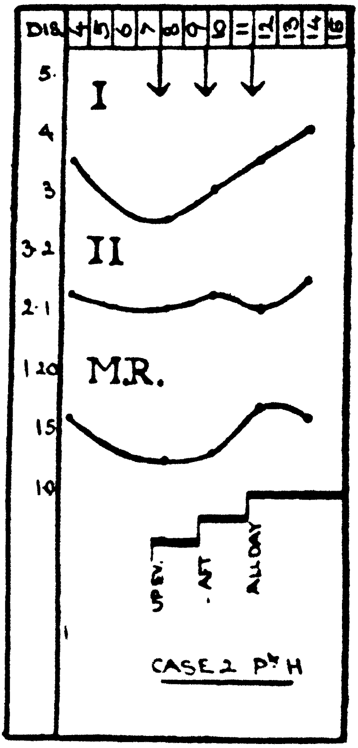

FIG. 5.

show the variation in the intensities of the first and second sound compared with the value of the ratio from day to day.

All the cases were clinically typical of mild influenza, and the history of one will serve for all.

CASE 1.—Pte. G., æt. 19. Ill two days before admission with shivering, pains in thighs and back, and a dry cough; he had some sneezing. With rest in bed and a mixture of creosote, potass. iodid. and tinct. camph. co., he rapidly recovered.

Two further diagrams (8 and 9) are shown of the only case with any complication. The history was as follows:—

CASE 7.—Private B., æt. 18. Taken ill the day before admission with headache, pain in the left side of the chest, and cough. On admission, the respirations were rapid and accompanied by groans;

the lips were parted and dry, but not cyanosed. On examination, slight impairment of percussion note was found over the lower left chest posteriorly with weaker breath sounds. The next day, signs of fluid in the chest were obvious; the sputum was copious, purulent, and bloodstained, but he had slept well and the pain was better. On the following day, considerable sweating occurred, and the whole condition took a rapid change for the better. The fluid had quite disappeared five days after admission.

THE NORMAL MYOCARDIAL RATIO.

So far the results entirely agree with those of previous investigations, except that in a number of cases the myocardial ratio did not rise to the normal 2:1 when measured recumbent, even though the patient appeared quite fit. The normal ratio in 20 men who had not been ill for some months and had been doing full duty until admitted with scabies or impetigo was therefore investigated. Two examinations in both the erect and recumbent positions were

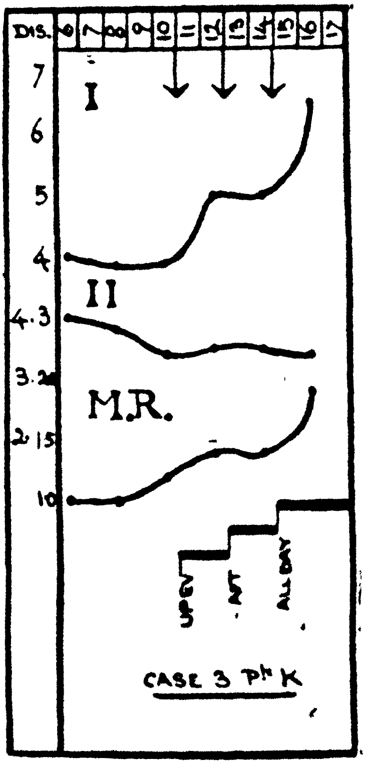

FIG. 6.

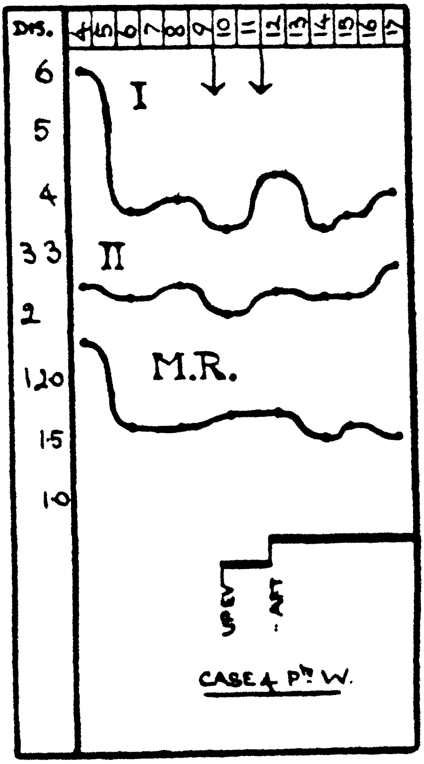

FIG. 7.

made on each patient on two separate days and the following readings obtained:—

	Average of First.	Average of Second.	Average M.R.
Erect - -	5·6	3·0	1·9
Recumbent -	3·7	2·6	1·4

That these results are typical, in spite of the small number examined, is seen by the slight divergence of the maxima and minima from the average M.R.

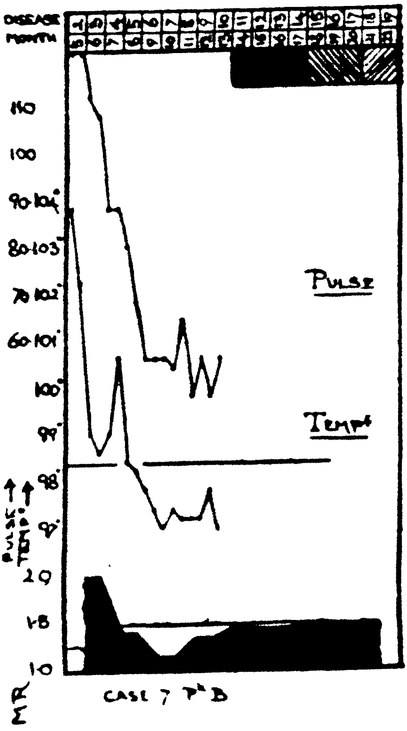

FIG. 8.

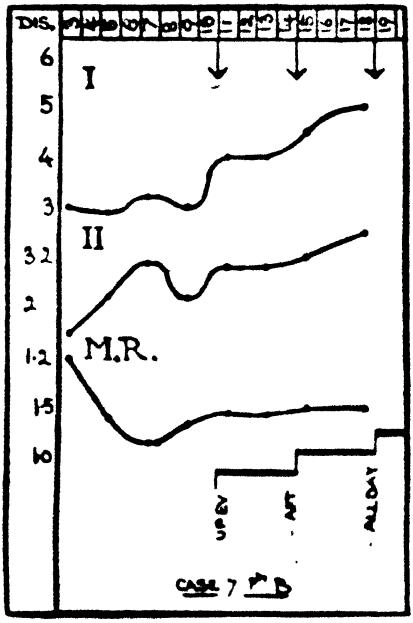

FIG. 9.

The actual intensities of the sounds naturally vary considerably owing to the different types of chest, etc.

	Max. 1st.	Min. 1st.	Max. 2nd.	Min. 2nd.	Max. M.R.	Min. M.R.
Erect -	7·0	4·5	3·75	2·3	2·2	1·7
Recumbent -	4·5	3·25	3·5	1·9	1·8	1·0

It appears, then, that the M.R. in the recumbent position is always lower than in the erect, and the normal figure for the former may provisionally be taken as 1·4. In the above series, 50 per cent. gave a M.R. of 1·3 or 1·4, and 75 per cent. 1·2 to 1·5 inclusive.

The average curve shown in the first diagram does not descend below this figure; but six out of 10 cases showed a lower figure at one time, although never below 1·0 (*cf.* Diagrams 4 and 6). The lowering of the ratio is therefore very little, and the point needing explanation is the high figure formerly considered normal found at the beginning of the illness and to a less extent in convalescence.

THE RATIO IN "IRRITABLE HEARTS."

The solution may be found in the study of patients suffering with "irritable hearts." Six cases of this condition in varying degree were examined, two of them being of considerable interest since they also had influenza. Each case, except one which was only examined once, was submitted to three or more tests, making 17 examinations in all. The results obtained were for the recumbent position:—

	Average.	Maximum.	Minimum.
1st	7·0	9·0	5·0
2nd	3·4	4·0	2·7
M.R.	2·1	2·9	1·4

(In 11 examinations the M.R. was 2·0 or more.)

The variation from recumbent to erect posture in these cases was not so decided as in normal cases, the average for the erect posture—in those examined—being 2·4 and the mean 2·2. The average was raised by the inclusion of one case with a ratio of 3·3.

The two cases mentioned above, who had influenza as well as D.A.H., are instructive. One had the latter condition on admission, and the other developed it while in hospital.

Case 18.—Private W. Suffered from D.A.H. in France in 1918, and was invalided back to England for it, going to Leeds

Cardiac Centre, where he was graded B3. He fell sick on the day before admission (April 6, 1919), with pain in the throat and chest and feeling sore all over. He had moderate fever, slight coughing, and some sneezing. With the usual remedies, he quickly became convalescent, but he continued to suffer with pain in the chest and other symptoms of "irritable heart." The following readings were obtained with the differential stethoscope :—

Date.	First Sound.	Second Sound.	M.R.
April 7, 1919 - -	6·5	3·0	2·2
April 9, 1919 - -	8·5	3·25	2·6
April 11, 1919 - -	8·5	3·5	2·4
April 28, 1919 - -	6·0	4·0	1·5

All these were taken in the recumbent position; the corresponding figures for the erect position on the last date were :— 10 4·0 2·5. This case showed a high ratio for the first seven days of illness depending on a high first sound.

The second case differs from this in that he developed, or rather re-developed, the D.A.H. during the course of the illness.

Case 21.—Lance-Corporal W. Gave a history of having suffered with symptoms of D.A.H. 18 months ago, for which he was discharged from the Navy in 1918. He enlisted in the Army later, and has been free from symptoms up to the present, except on exertion. Admitted to hospital on April 13, 1919, he complained of having been ill for three weeks off and on, but that he was much worse the last two days with cough, shivering, headache, running from the nose, and pains all over. His influenzal symptoms ran the usual course, except that on the 16th his pharynx was considerably congested, and that two days later (18th) he complained of pain in the chest and knees. The apex-beat was then noted to be very forcible and somewhat diffuse; this condition, with breathlessness on exertion after he got up, continued up to date of discharge. The readings obtained in this case were :—

Date.	First Sound.	Second Sound.	M.R.
April 14, 1919 - -	4·75	2·2	2·1
April 16, 1919 - -	4·75	3·35	1·4
April 18, 1919 - -	8·0	4·0	2·0
April 21, 1919 - -	6·5	4·0	1·6
April 23, 1919 - -	7·0	4·0	1·7
April 25, 1919 - -	9·0	4·5	2·0
April 28, 1919 - -	5·0	3·5	1·4

In this case, note the abrupt rise in the intensity of the first sound associated with symptoms of "irritable heart." With an intensity of more than 4·5 in the recumbent, a condition of irritability may be suspected.

Applying these results to the interpretation of the high ratio found during fever, the occurrence of over-action of the heart-muscle is very probable. In some cases in convalescence a similar condition was met with, the intensity of the first sound being unduly increased in the recumbent position; the inclusion of these cases in the preparation of the composite diagram (1) has tended to raise the curves from the fifteenth to the twentieth day.

The following observation is interesting in this connection. Lewis puts down the origin of most cases of irritable heart to infectious disease. Histories of onset after such illnesses were obtained in 33 per cent. of his cases, and he states,[8] "that infectious disease is probably responsible, in part or in whole, in a considerable number of the remaining 67 per cent." In the table given (3, page 71), only 9 per cent. of all cases of D.A.H. had suffered with influenza, as compared with 42 per cent. who had had rheumatic fever or chorea; but the former disease has been comparatively rare in the young until the past epidemic, and in future it may figure as a more prominent cause of this condition.

As regards time of onset in convalescence and relations to length of stay in bed, the following figures were obtained in the 10 cases studied intensively :—

Cases developing irritable hearts - - - - 4
Cases not so affected - - - - - - 6

Case.	Day of disease ad-mitted.	No. of days fever ob-served.	Day of disease temp. normal.	Days between temp normal and getting up.	Day of disease first up	Days after getting up first signs of D A.H.	Day of disease.	Day of admis-sion	Days after last day of fever.
1	5	3	8	3	11	1	12	7	5
2	2	3	5	1	6	5	11	10	7
3	4	1	5	7	12	6	18	15	14
4	2	3	5	6	11	7	18	17	14

Those who had no signs of D.A.H. gave the following results :—

Case.	Day of disease admitted.	No. of days fever observed.	Day of disease temp. normal.	Days between temp. normal and getting up.	Day of disease first up.	Days after first getting up of last observation.	Day of disease.	Day of admission.	Days after last day of fever.
1	3	5	8	2	10	5	15	13	8
2	1	4	5	3	8	6	14	14	10
3	3	4	7	3	10	7	17	15	11
4	1	1	2	3	5	7	12	12	11
5	2	1	3	4	7	8	15	14	13
6	4	2	6	5	11	6	17	14	12

All the illnesses were moderate except Case 4 (Case 7 of whole series) of the D.A.H. series, who developed a pleural effusion.

Marris[9] showed that in trench fever, which seems clinically very closely allied to influenza, vasomotor tachycardia or D.A.H. is very apt to develop. He found that keeping the patient in bed for eight clear days after the temperature had become normal, materially reduced the incidence of this condition, and rendered it less resistant to treatment.

Whether the same results may be obtained in influenza has not been attempted, owing to the limited number of cases seen and the fact that the writer was unable to read the above paper until the epidemic was over. The whole question of occurrence and possibility of permanent D.A.H. resulting from influenza is in need of investigation.

The cause of the D.A.H. may be the same as that put forward by Lewis,[10] who suggests that the syndrome may be due to a lack of buffer salts, which has been found to occur by Barcroft, and which explains the cardinal symptom of breathlessness. This can only be proved by estimation of these salts in the course of the illness and convalescence.

CONCLUSIONS.

It can be said, then, that:—

1. The myocardial ratio cannot be considered abnormal, when measured in the recumbent position, unless it falls below 1·2.

2. There is always lowering of the ratio on changing from the erect to the recumbent position.

3. The amount of this lowering is less in the case of irritable hearts.

4. If a ratio of more than 1·6 is found in a recumbent patient, especially if associated with a first sound of intensity more than 4·5, over-action of the heart should be suspected.

5. In mild, uncomplicated influenza, little change takes place in the ratio, and not enough to support belief in the direct toxic action of the influenza bacillus on the heart muscle as a common occurrence. It is probable that fainting, breathlessness, heart pain, etc.—all symptoms of D.A.H.—have been mistaken for symptoms of myocardial weakness.

6. Influenza may be one of the causes of permanent D.A.H., which appears as a *temporary* condition early and sometimes late in the disease.

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Estimations of Sugar in the Blood in the Diagnosis and Treatment of Diabetes.

By P. J. CAMMIDGE, M.D., D.P.H.

THE investigation of the chemistry of the blood has made rapid strides in the past decade, and many valuable indications have been obtained regarding the disturbances which occur in the abnormal production and elimination of waste products by the body.

So long as comparatively large quantities of blood were required for such investigations their usefulness for purposes of diagnosis and treatment was very limited, but with the micro-methods now available many of these findings can be applied as a clinical routine, with no more discomfort to the patient than is caused by a prick with a needle. Alterations in the chemistry of the body may consequently be investigated with a precision which was previously impossible, and by comparing the results of analyses of the urine with the data furnished by blood-examinations, disorders of metabolism can be studied from a new point of view, and a reliable picture of the functional activity of the kidneys may be secured. The two diseases, in which chemical analysis of the blood is particularly useful, are nephritis and diabetes; determinations of the end-products of nitrogenous metabolism, and their relation to each other, by this means being the most certain guide we have to the diagnosis and prognosis of the former, while estimations of the sugar-content of the blood are proving to be an essential check in the diagnosis and treatment of the latter, as I propose to show.

Before discussing the sugar-content of the blood in diabetes, it is necessary that we should consider briefly the methods by which the estimation may be made and the figures given by healthy people under various conditions. Up to a few years ago clinical estimations of the percentage of sugar in the blood were usually made by Bang's method.[1] Although only a few drops of blood are required, the use of a torsion balance is necessary and great care is needed to obtain accurate results, so that in practice it can only be carried out in a properly equipped laboratory by an observer trained in chemical technique. In 1915, Lewis and Benedict[2] described a new process, which requires no very special apparatus and is so simple that it can be carried out in a quarter of an hour by anyone familiar with ordinary chemical procedures. Their method is an adaptation of Braun's picric acid test for sugar, and depends upon the fact that when a solution of sugar is heated with picric acid and an alkali a deep red colour, due to the formation of picramic acid, develops, and, as the depth of the colour is proportional to the amount of sugar present, the percentage can be determined by comparison with a standard solution.

Lewis and Benedict advise the use of 2·0 cc. of blood, but I find that, with the modification recently described by Benedict,[3] satisfactory results for clinical purposes can be obtained with 0·2 cc. This amount of blood is mixed with twice its volume of water, and then gradually made up to 2·5 cc. with constant shaking, by adding a specially prepared solution of sodium picrate-picric acid. The resulting precipitate of protein is filtered off and 1·0 cc. of the clear filtrate placed in a small test-tube with a 10·0 cc. mark. After adding 0·1 cc. of a saturated solution of sodium carbonate, the tube is plugged with cotton wool and heated in a boiling water bath for ten minutes. The tube is then cooled and its contents made up to the 10·0 cc. mark with water. The depth of colour of the resulting fluid is compared with a standard solution of picramic acid, or a 0·2 per cent. solution of pure dextrose treated in exactly the same way, in some form of colorimeter.*

Another method which requires only 0·2 cc. of blood was described by me in 1917.[4] It takes a little more time than the picric acid process, but gives more accurate results when carried out with proper precautions, and, moreover, avoids the use of a colorimeter, since it depends upon the titration, with a standard solution of sodium thiosulphate, of the amount of standard iodine left uncombined after adding the copper chloride formed when a protein-free filtrate of the blood has been boiled with an alkaline solution of copper and treated with hydrochloric acid. Recently Folin and Wu[5] have published a new process for estimating sugar in the blood which appears to be suitable for clinical work.

In the modification I have used, 0·2 cc. of blood is mixed with 3·0 cc. of water. The protein is precipitated and removed by adding 0·4 cc. of the sodium tungstate solution (half strength) described by Folin and Wu, and 0·4 cc. of one-third normal sulphuric acid, heating in a boiling water bath for two or three minutes, and then filtering. Two test tubes with a 12·5 cc. mark are now taken and into one is introduced 2·0 cc. of the clear filtrate and into the other 1·0 cc. of a 0·1 per cent. solution of pure dextrose, which is used as the standard. The contents of the tubes are treated in exactly the same way. To each is added 1·0 cc. of a special copper solution and they are heated in a boiling water bath for six minutes, immediately 0·5 cc. of a molybdate-tungstate mixture is added and the contents of each tube well mixed. They are now cooled and, after 2·5 cc. of a saturated solution of sodium carbonate have been introduced, the contents of the tubes are made up to the 12·5 cc. mark with water. After standing for five minutes, the resulting blue solutions are compared in a colorimeter and. from the readings obtained, the percentage of sugar in the unknown is worked out.

As an essential preliminary for any diagnostic procedure, it is needful to establish normal values. Numerous observations have shown that the blood does not contain a fixed amount of sugar, but has a fairly wide range, varying between 0·06 and 0·14 per cent., with an average of 0·10 per cent. Unfortunately, the average value is often the only figure quoted by writers on diabetes, but it is of little or no importance for purposes of diagnosis, what is necessary is that we should realize that 0·14 per cent., or even 0·15 per cent., of sugar in the blood is quite consistent, under certain conditions, with perfect health. The chief conditions which determine the proportion of sugar in the blood are the nature of the food, and the time after a meal the determination is made.

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* A simple form of colorimeter, suitable for this and other colorimetric work, has been made for me by Messrs. Hawksley and Sons, 357, Oxford Street W. 1.

In a normal healthy individual, a specimen of blood, taken in the morning, after the night fast and before any food has been consumed, usually shows from 0·06 to 0·08 per cent. of sugar. As digestion and absorption progress, the sugar-content of the blood rises until a maximum, not exceeding 0·14 to 0·15 per cent. is reached, the exact interval of time and percentage attained depending upon the composition of the meal and the functional activity of the digestive tract. If no further food is eaten, the percentage of sugar in the blood gradually sinks until the fasting level is again reached, but if another meal is taken before this point is arrived at, a fresh rise occurs, and a similar curve to that previously described follows. The maximum level attained never exceeds 0·14 per cent., or at most 0·15 per cent., no matter what the amount or character of the food may be.

When an ordinary meal, containing a mixture of carbohydrates, protein, and fat is taken, the highest point in the blood-sugar curve is usually reached in about three hours; if there is little fat the time may be shorter, a large amount of fat may prolong it. Proteins and fats alone do not appreciably increase the percentage of sugar in the blood. All forms of carbohydrate cause a rise, the only difference between sugar and starches being the rate at which the increase takes place, and the time that elapses before the fasting level is reached again. Sugar, being quickly absorbed, brings about a rapid increase, followed by an almost equally rapid fall.

whereas the more slowly digested and absorbed starches give a curve of similar height but spread over a longer period in both its rise and fall.

The sugar-content of the blood in diabetes varies with the nature of the food and the time after meals in the same way as in health. The maximum percentage reached is usually higher, however, and a longer period always elapses before the effect produced by the ingestion of carbohydrates passes off; that is to say, the blood-sugar curve in diabetes is similar in form to the normal, but it has a wider time relation and in typical cases attains a greater height. In mild forms of the disease, the percentage of sugar in the blood may be within normal limits in the fasting condition, and even when starchy foods, such as bread, are taken it may not rise beyond 0·14 per cent. or 0·15 per cent., but the return to the previous level is invariably delayed, showing that the powers of the organism for disposing of sugar are defective.

When a patient of this type is given a test-meal of dextrose (glucose) and the blood is examined at frequent intervals the variation from the normal becomes more apparent. Instead of a maximum of 0·14 to 0·15 per cent. being reached in an hour, as in health, the proportion of sugar in the blood continues to rise for another half-hour or more, and percentages of 0·20, or higher, are registered. The fall to the fasting level, too, instead of being complete in one-and-a-half to two hours from the commencement of the experiment, occupies four or five hours.

With increasing severity of the defect in carbohydrate metabolism, the deviations from the normal in the time and space relations of the sugar curve become more and more pronounced, until, eventually, a permanent hyperglycæmia is established, and the percentage of sugar in the blood never drops within normal limits, even after the night fast. A meal containing only a moderate amount of starch will then send the blood-sugar up to 0·40 or 0·50 per cent., and in one case I found 0·80 per cent. three hours after 50 grams of carbohydrate had been taken in the form of oatmeal and bread. Test meals of sugar are obviously unnecessary in such cases, and it is not wise to use them, but in one instance in which I tried the experiment of giving 50 grams of dextrose it resulted in a rise from 0·17 to 0·48 per cent. in the blood-sugar in two hours, and 12 hours later the former percentage had not been reached again although the patient had fasted meanwhile.

It follows from what has been said that blood-sugar determinations to serve their greatest usefulness should be performed both before and at a known interval after an ordinary meal containing carbohydrate, and that in doubtful cases the effects of a test meal of dextrose should also be observed. Sansum and Woodyatt have called attention to the inaccuracies of the usual clinical method of tolerance testing for sugar, and suggested a more accurate procedure, but as it requires the use of expensive apparatus it is not likely to be generally adopted. A more convenient, and yet sufficiently accurate, procedure has been suggested by Janney and Isaacson.[6]

The patient fasts from 7 p.m. one day, until the test is completed the next morning. Meanwhile his body-weight, naked, is ascertained, and a solution of dextrose is prepared by dissolving in water a weight of the sugar equivalent to 1·75 grams per kilogram of this weight, allowing 2·5 cc of water for each gram of dextrose used. At an early morning hour, the percentage of sugar in the blood is determined. As soon as convenient thereafter the patient drinks the prepared dextrose solution, and, if he wishes, washes out his mouth with a little of the water reserved from the measured quantity for the purpose. Exactly half-an-hour after the sugar has been taken, a specimen of blood is withdrawn for a second sugar determination, a third is examined half-an-hour later, and so on every half-hour for two hours, or as long as may be deemed necessary.

In a normal person the difference between the percentage of sugar in the blood two hours after the sugar has been swallowed and when fasting should not exceed 0·01 per cent. If the patient's carbohydrate tolerance is diminished, the determination made at the end of two hours will show hyperglycæmia, and the fall to the fasting level will be delayed according to the degree of the defect. For diagnostic purposes, it is advisable that a starch containing meal of definite composition should also be taken.

The test meal I usually employ contains one gram of carbohydrate, two-thirds of a gram of fat, and one-third of a gram of protein per kilogram of body weight. For a man of 70 kilos (11 stones) this can be made up with ¾ oz. of oatmeal (dry weight) made into porridge, 3½ oz. white bread, 3 oz. of milk, 2 oz. of average bacon (weighed raw), and one egg, with tea or pure coffee as desired. Such a meal contains about 70 grams of carbohydrate, of which about 4 grams are lactose, 50 grams of fat, and 25 grams of protein, and is capable of yielding about 90 grams of sugar, when the sugar formed from protein is taken into account. The patient fasts from 7 p.m. in the evening, as in the sugar test, and a specimen of blood is taken next morning, fasting. He is then given the test breakfast, and subsequently the sugar-content of the blood is determined at hourly intervals for five or six hours.

In a normal person a maximum percentage of sugar should be found at about the third hour after the meal, and it should not be higher than 0·15 per cent. Any excess over this figure, and a delay beyond the fifth hour in the fall to 0·10 per cent. show that carbohydrate metabolism is defective, and that the patient must, at least, be classed as a potential diabetic.

The urine has been for so long the guide to diagnosis and treatment in diabetes, that it is important to establish a relationship between the sugar-content of the urine and blood if possible. The threshold point, that is the percentage beyond which an increase in the proportion of sugar in the blood is followed by the appearance of sugar in the urine, has been the subject of much debate. One of the difficulties in fixing a definite threshold value is to decide what constitutes a normal amount of sugar in the urine, for it is now generally agreed that all urine contains a small percentage of sugar. Recent observations, with improved quantitative methods, have shown that the amount is dependent upon the nature and quantity of the food; thus, Benedict[7] found on an ordinary mixed diet an average excretion of 1 gram a day, on a low carbohydrate diet 0·75 gram, and on a high carbohydrate diet 1·5 grams in the 24 hours. When the course of the sugar excretion was investigated at two-hourly intervals, it was seen to be at a minimum after the night fast, for the two hours following breakfast it was about 300 per cent. higher than for the previous fasting hours, then it fell and continued to fall until a fresh rise occurred after the next meal.

It is important to note that on a diet rich in carbohydrate as much as 0·6 per cent. of sugar was found in some specimens of urine collected during the two hours after breakfast. As this percentage will give a definite reaction with sensitive tests, such as Benedict's, a mistaken diagnosis of diabetes might be made if such a sample happened to be examined, for life insurance or other purposes, without being controlled by analysis of a 24 hours' collection, or a determination of the percentage of sugar in the blood. Unfortunately, no observations on the sugar-content of the blood appear to have been made in Benedict's cases. My experiments with normal people suggest that the output of sugar in the urine follows a curve similar to that described by the percentage of sugar in the blood, only it is delayed.

In one normal individual, for example, estimations of the blood-sugar at hourly intervals and determinations of the total amount of sugar excreted in the urine during the previous hour give the following results :—

—	Fasting.	Breakfast.	1 hr.	2 hrs	3 hrs	4 hrs	5 hrs	6 hrs
Per cent of sugar in the blood - -	0·07	Porridge, bread and butter, egg, bacon, coffee, milk, sugar.	0·10	0·13	0·15	0·135	0·10	0·10
Total sugar in urine in grams - - -	0·02		0·02	0·03	0·04	0·05	0·04	0·04

Others have shown similar figures. It would seem, therefore, that there is no absolute threshold point for the appearance of sugar in the urine, and that the difference between normal and the diabetic urine is chiefly one of degree.

A study of over 700 cases of diabetes, which have come under my care, has proved that there is no constant blood-sugar level for the appearance of sugar in the urine in quantities recognizable by ordinary tests, also, that there is no definite relationship between the percentage of sugar in the blood and either the percentage or total amount of sugar excreted by the kidneys. Patients with a permanently high blood-sugar may pass comparatively little sugar in their urine, while, in some instances, a normal, or even a subnormal, blood-sugar curve may be associated with frank glycosuria. In either condition, examination of the urine alone does not give a correct picture of the case, and, if it is not checked by blood-sugar estimations under controlled conditions, may readily lead to mistakes in diagnosis and treatment. As a rule, young diabetics have a lower threshold point for clinical glycosuria than those of middle age, and the threshold rises with advancing years. It is, therefore, important that the presence of even small amounts of sugar in the urine of persons of middle age should not be dismissed as of little significance, unless a series of blood-tests have shown that their tolerance for carbohydrates is not seriously defective. A more general adoption of this plan would save much unnecessary suffering in the shape of complications, and avoid many unexpected deaths from diabetic coma.

Hyperglycæmia may exist without clinical glycosuria, that is with an insufficient percentage of sugar in the urine to give the ordinary tests for sugar. Such a condition is not uncommonly the explanation of recurrent attacks of boils, carbuncles, sciatica, neuritis, eczema, etc., which do not respond to ordinary methods of treatment. When the true cause is discovered by examination of the blood, and the diet is arranged to bring down its sugar-content to normal, a rapid improvement follows. Hyperglycæmia without glycosuria is also common in obese people, some 25 per cent. showing an abnormally high percentage of sugar in their blood. In this connection, it is interesting to see that Joslin[8] found decided obesity to have preceded the onset of diabetes in over 40 per cent. of his cases. Occasionally, a high percentage of sugar in the blood may be associated with the passage of an excessive amount of urine, but without glycosuria. In such cases, a diagnosis of diabetes insipidus may be made unless the blood is investigated.

The reverse condition, glycosuria with a normal or subnormal percentage of sugar in the blood, is not as uncommon as is generally supposed. Cases of this description may be divided into two main classes, first, those in which a test-meal of starch does not cause hyperglycæmia, but the subsequent return of the blood sugar to the previous fasting level is abnormally delayed; second, those in which the blood-sugar curve follows an entirely normal course. The administration of a test-meal of dextrose to the former causes more or less hyperglycæmia, thus confirming the existence of a latent diabetic tendency, whereas with the latter the percentage of sugar in the blood does not rise above the normal level, and, indeed, often fails to reach it. The one may be termed "latent diabetes," and probably corresponds in part to the class of case described by Reicher[9] under that name, the other is what has been termed "renal diabetes" or "renal glycuresis."

My own observations suggest that many cases of latent diabetes are essentially hepatic in origin, and that so long as the patient avoids sugar and foods containing sugar as such, he may take any starchy food in moderation without harm, provided that the protein and fat content of the diet are also controlled. It is advisable, however, that these patients should have their blood examined at intervals, in order that any further failure in the mechanism of carbohydrate metabolism may be quickly recognized and dealt with. Renal diabetes is probably a congenital abnormality, and depends upon an abnormal permeability of the kidneys for sugar. Restriction of the diet is unnecessary and appears to do more harm than good, for it renders the patient less able to withstand prolonged physical exertion. I have met with six cases, and the only complaint made by the patient in each instance was that he was easily tired. Two of these cases are particularly interesting, for they were father and daughter, and, so far as I am aware, heredity has not been reported previously in this condition.

The father, a man aged 32 years, had been treated for diabetes for many years before I saw him. He then stated that sugar had been first discovered in his urine accidentally when he was 16, and no treatment he had ever undergone had rendered him sugar free. He had never had any of the usual symptoms of diabetes, but he had noticed, even when a boy at school, that he was more easily fatigued than others of his age. Analysis of his urine showed 5 grams of sugar for the 24 hours, but no other abnormality. When his blood was investigated it gave 0·06 per cent. of sugar three hours after an ordinary mixed meal containing 75 grams of carbohydrates. I took him into a nursing home for observation, and, although the carbohydrate content of his diet was repeatedly varied between 30 and 190 grams a day, the sugar in his urine never fell below 3 grams nor rose above 6 grams for the 24 hours. His blood-sugar ranged from 0·06 per cent. to 0·09 per cent. three hours after meals, and fell to 0·05 per cent., in the morning, fasting. When he was given a test-meal of dextrose (80 grams) his blood-sugar rose from 0·05 per cent. to 0·07 per cent. in the first half-hour, at the end of one hour it stood at 0·11 per cent., and after that it fell again until, at the end of the second hour, it had dropped to 0·06 per cent. I have watched this case for over a year now and in spite of his taking an ordinary full diet meanwhile, there has been no change in the amount of sugar passed in the urine and his blood-sugar has averaged 0·07 per cent.

The child of this patient, a girl aged 6 years, was found to be passing sugar in her urine at the age of four. She was treated for diabetes, but, in spite of strict dieting, never lost the glycosuria. I found she was passing rather more sugar than her father, especially in relation to her weight, and her blood-sugar stood at a slightly higher level. With changes in the carbohydrate content of her diet, between 50 and 97 grams a day, the output of sugar in her urine ranged from 3 to 7 grams for the 24 hours. Blood-sugar examinations showed an almost constant maximum level of 0·08 per cent. three hours after meals, with a rise to 0·10 per cent. on one occasion. Fasting, the blood-sugar fell to 0·06 per cent., and there was no abnormal delay in reaching that level after a carbohydrate meal. A test-meal of dextrose (26 grams) resulted in a maximum blood-sugar of 0·13 per cent in half-an-hour, with a return to 0·07 per cent. in two hours.

A comparatively low blood-sugar-content is usually a good sign in ordinary frank diabetes, even when a large amount of sugar is being passed in the urine. Such patients generally lose their glycosuria rapidly when properly treated and often regain a comparatively high carbohydrate tolerance.

For example, a patient, who came under my care a couple of years ago, passed 220 grams (over 8 per cent.) of sugar on a diet containing 76 grams of carbohydrate on admission, but the maximum blood-sugar level three hours after food was never higher than 0·15 per cent. All sugar disappeared from the urine in five days, and at the same time the blood-sugar came down to 0·08 per cent. Ten days later, she was able to take a diet containing 65 grams of carbohydrate without any trace of sugar appearing in the urine, and with a maximum blood-sugar of 0·12 per cent. Subsequently, the patient's tolerance has increased still further and she is now taking 80 grams of carbohydrate a day, 4 grams more than had previously caused the excretion of 220 grams of sugar in the urine.

Determinations of the sugar-content of the blood are quite as important in the treatment as in the diagnosis and prognosis of diabetes. It is obviously impossible to treat cases of hyperglycæmia with no glycosuria on the results of analyses of the urine alone, and in this class of case one is bound to rely on the results of examinations of the blood. But blood-sugar estimations are of almost equal value in other forms of diabetes to check the indications given by the urine. As we have seen, there is no constant relation between the sugar-content of the blood and the output of sugar in the urine, so that it not infrequently happens that, when the urine of a diabetic has been rendered "sugar-free" by fasting, or other means, the blood-sugar still stands at an abnormally high level. If the indications given by the urine alone are then relied upon, and the carbohydrate content of the diet is increased, there may be no immediate return of the glycosuria, but the downward progress of the case will continue, and an ultimate recurrence is certain. If, on the other hand, the warning given by the high blood-sugar is heeded, and a low diet, or fasting, is continued until it is reduced to at least 0·10 per cent., and subsequent increases in the diet are regulated by their effect on the blood rather than on the urine, the progress of the disease may be stayed, and the patient may be able to tolerate ultimately a much more liberal diet without hyperglycæmia or glycosuria than would otherwise be possible. Many cases which are apparently intractable and show progressive loss of tolerance, when the treatment is based solely upon examination of the urine, improve to a remarkable extent when the diet is controlled by frequent analyses of the blood. One of the most important factors in determining the permeability of the kidneys to sugar in diabetes appears to be the fat-content of the diet. It has been my experience that nearly all cases who have for some time taken an abundance of fat, as in the regulation "carbohydrate-free" diet, lose the sugar from their urine some time before the percentage of sugar in their blood falls within normal limits when they are treated by modern methods, and that the interval varies directly with the amount of fat they have been in the habit of taking.

Thus one patient who had been on a "carbohydrate-free" diet containing a large amount of fat in the form of cream, eggs, so-called diabetic biscuits, etc., for four years before she came under my care, passed 55 grams of sugar in her urine on a test-diet containing 75 grams of carbohydrate, 80 grams of fat, and 70 grams of protein, and her blood-sugar stood at 0·40 per cent. Four days later, the output of sugar in the urine had fallen to 2 grams as a result of a reduction of the carbohydrate to 25 grams and the fat to 20 grams, but the percentage of sugar in her blood remained unchanged. All fat was then eliminated from her diet as far as possible, with the result that next day no sugar could be found in the urine and the blood-sugar had dropped to 0·30 per cent. On the same diet, the blood-sugar had fallen to 0·24 per cent. next day, to 0·18 per cent. 24 hours later, to 0·12 per cent. in another 24 hours, and at the end of two days more to 0·09 per cent. The diet was then gradually increased again, and eventually was worked up to 56 grams of carbohydrate without a return of the glycosuria or any hyperglycæmia, although a similar amount of starchy food on the second day of the treatment had resulted in the excretion of 38 grams of sugar in the urine, with 0·40 per cent. of blood-sugar.

Permanent success in the treatment of diabetes can only be secured by keeping the blood-sugar curve within normal limits, even though more severe restriction of the diet may be necessary to attain this end than is required to keep the urine sugar-free. Although 0·15 per cent. may be regarded as the maximum normal blood sugar level at the height of digestion and absorption, it is safer, if the diet of a diabetic can be so arranged, that the highest point reached does not exceed 0·13 per cent. As long as this figure is maintained the outlook is good, but a progressively rising blood-sugar is a serious prognostic sign, and suggests the need for an immediate revision of the diet, even when the urine is free from sugar. In fact, a high blood-sugar level with no glycosuria is more serious than a low percentage of sugar in the blood with moderate glycosuria, for a pound of sugar in the urine has no greater practical significance than a pound in a grocer's shop, unless it indicates an excess in the blood.

A progressively increasing blood-sugar level without glycosuria is often due to the diet containing too high a proportion of protein or fat, especially the latter. When the fat is reduced, even though no other alteration is made in the diet, the percentage of sugar in the blood will often come down, and the tolerance of the patient for carbohydrate will be found to have increased; but should the warning given by the blood-sugar be disregarded and the diet be left unchanged, an eventual return of the glycosuria is certain to occur, although it may be delayed for weeks or even for months. Cutting down the carbohydrate to suit the apparently reduced tolerance of the patient in such a case may be of temporary benefit, but if the fat and protein are left at their previous level the blood-sugar will again slowly mount up, with recurrence of the glycosuria and another apparent fall in the carbohydrate tolerance later on. Further re-adjustment of the carbohydrate will probably be followed by a temporary control of the glycosuria and hyperglycæmia, but the downward progress of the case will continue, and a fatal termination is inevitable so long as an unbalanced diet is persisted in.

When the treatment is controlled by blood analyses, and the effect of fat on the permeability of the kidneys for sugar is understood and acted on, the whole aspect of the case changes and an unfavourable is changed into a favourable prognosis. Such seemingly hopeless cases of progressive diabetes are particularly common among young people, and the explanation is usually unsuitable treatment, not some inherent malignancy in the disease. A rising blood-sugar-content in elderly people is frequently dependent upon the diet containing too high a proportion of protein. Restriction of the protein and fat, with an increase in the carbohydrate, often results in an improvement in the general condition, and is followed by a drop in the sugar-content of the urine and in the percentage of sugar in the blood.

Too hasty a diagnosis of diabetes should not be made from the presence of an excess of sugar in the blood nor even from an abnormal blood-sugar curve after a test-meal of sugar, for other diseases may be associated with hyperglycæmia. In the later stages of nephritis, for example, the percentage of sugar in the blood is usually high, often equalling the amount met with in severe diabetes when uræmia is imminent, but the blood-picture is one of complete metabolic failure, and the end-products of nitrogen metabolism are correspondingly increased. In the early stages of the disease the blood-sugar is usually normal, failure in nitrogen metabolism often preceding by some months a rise in the blood-sugar. As the pathological changes in the kidneys progress, and these organs become more and more impermeable to sugar, it is held back and accumulates in the blood in increasing quantities. A high blood-sugar, therefore, is of serious omen in nephritis, and indicates a speedy fatal termination. In some instances a small amount of sugar may leak through the kidneys and, being found in the urine, lead to a diagnosis of diabetes, particularly if an examination of the blood is made and a high sugar level is discovered. A diabetic diet is unnecessary in these cases, and is indeed harmful if it is of the "carbohydrate-free" type, for restriction of the carbohydrate intake has no appreciable effect on the level of the sugar in the blood and a high proportion of protein is contra-indicated by the condition of the kidneys.

Some excess of sugar in the blood is usually found in patients suffering from cardio-vascular diseases with high blood-pressure, even when there is little or no indication of renal disturbance. Here again control of the protein intake, rather than of the carbohydrate allowance, is the correct treatment. Carcinoma is another condition in which it is said that there is often moderate hyperglycæmia, ranging up to 0·1 per cent. according to Williams and Humphreys.[10]

In a recently published paper, Rhodenburg, Bernhard, and Krehbiel[11] state that the administration of a test-meal of dextrose in cases of malignant disease always results in an abnormal blood-sugar curve, and that by this means cancer may be differentiated from other conditions with which it may be confused. They found a rapid rise to 0·18 per cent., or even 0·20 per cent., occurring in 45 minutes after the sugar had been taken. At the end of two hours there was either no change, or a further rise to 0·288 per cent., and, in one instance, to 0·35 per cent., had occurred. The subsequent fall in the curve is gradual, and does not reach the normal level until the lapse of three or four hours after the ingestion of the sugar.

As yet comparatively few cases have been investigated, but if their results are confirmed it will be a valuable help in diagnosis. The explanation of the high blood-sugar curve in malignant disease is not clear, but it is possible that it may be due to faulty functioning of the endocrine glands, for a similar hyperglycæmic response, followed by a delayed fall to normal, after a test-meal of sugar has been observed in hyperthyroidism and exophthalmic goitre. In cretinism, myxœdema, acromegaly, and muscular dystrophy there is also a delayed blood-sugar curve, but, according to Janney and Isaacson[12] it is associated with hypoglycæmia.

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Syphilis of the Throat, Nose, and Ear; its Diagnosis and Treatment.

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IN the practice of the oto-laryngologist, syphilis makes its appearance almost daily, and its detection is often a difficult matter. Hence a brief description of the commoner lesions affecting the throat, nose, and ear may prove useful to those practitioners whose opportunities for hospital study are few, and who have little time to consult the voluminous literature on the subject.

SYPHILIS OF THE PHARYNX.

(a) *Primary. The Chancre.*—In his remarkable work on *Syphilis of the Innocent*, Bulkley states that syphilis is not essentially a venereal disease, and that, in at least 10 per cent. of the cases, infection enters by some channel other than the genital organs.

The statistics of Fournier show that, in 10,000 cases of primary syphilis, the seat of the chancre was some part other than the genital region in 850 cases. Of those 850 cases of extragenital chancre, the lesion was in the region of the head in 75 per cent., and mostly in or about the mouth and throat. Among Fournier's cases 50 per cent. of all extragenital chancres were on the lips.

As regards the remaining 50 per cent., the finger, the tongue, and the tonsil were affected with equal frequency, each contributing about 17 per cent. to the sum total.

Chancre of the tonsil, though comparatively rare, is frequently missed or wrongly diagnosed. It may be mistaken for tonsillitis, diphtheria, carcinoma, or gumma. The important features are—

1. The cartilaginous induration on palpation with the gloved finger;
2. The fact that only one tonsil is affected;
3. The presence of enlarged cervical glands; and
4. The persistence of the lesion for several weeks.

Discovery of the spirochæta pallida may assist diagnosis, but it is well to remember that other spirochætes (buccalis, dentium, etc.) are common in all ulcerative conditions of the mouth.

(b) *Secondary. The Mucous Patch.*—This is the commonest of all syphilitic lesions, and is certainly the most contagious. Its favourite sites are, in order of frequency, the tonsil and the faucial pillars, the tongue, and the inner aspect of the lips. As a rule, the mucous patch appears as a bluish-grey streak, like a patch of paint, on tonsil and palate, and it is often symmetrical and frequently multiple. Although secondary syphilitic ulceration may attack the mouth, the typical mucous patch is an erosion rather than an actual ulceration. The terms "opaline" and "snail track" are often applied to it, but the usual colour is dirty grey. In a case which I saw recently, the entire surface of soft palate was involved.

The presence of enlarged glands and skin affections may in some cases facilitate diagnosis.

Though usually classed as a "secondary" lesion, appearing in the second or third month after infection, it may be seen at any stage of the disease, even many years after infection. The classification by Ricord into primary, secondary, and tertiary syphilis must not be too rigidly interpreted.

Prompt recognition of the mucous patch is highly important, for it teems with spirochætes and is a most fruitful source of infection.

The tonsils are frequently enlarged in secondary syphilis, and a reddening of the palate and fauces may appear (syphilitic erythema), both of which phenomena may assist the diagnosis.

(c) *Tertiary. The Gumma.*—This lesion seldom appears until after the first two years of the disease, and often only after the lapse of many years. It is a hard, purplish swelling, which attacks the palate, the posterior pharyngeal wall, or, less frequently, the tonsil. The swelling soon breaks down at its centre, becoming an ulcer with a greenish yellow base, red indurated edges, and a "punched out" appearance which is often quite characteristic.

The palate may be perforated, with considerable destruction of tissue, and this is especially true of hereditary syphilis, in which tertiary phenomena appear at about the age of puberty. When the posterior wall of the pharynx is also involved, the soft palate may adhere to it, shutting off the nasopharynx during the healing process with a degree of cicatricial stenosis unequalled by any other disease.

Tertiary ulceration of the tonsil may resemble Vincent's angina. In both, the Wassermann reaction may be positive, but the discovery of the spirochæta fetida and fusiform bacillus may assist in the diagnosis of Vincent's angina.

Gummatous ulceration may also simulate carcinoma, though cancer is usually characterized by the more advanced age of the patient and greater pain. Histological examination may decide the question. Lupus has a finely nodular appearance, and its lesions are usually present on the skin as well as on the mucous membrane. The destructive effects of lupus are much more slowly attained than those of syphilis.

SYPHILIS OF THE NOSE.

Primary lesions of the nose are rare, although, in a number of cases, the naso-pharynx has been infected by the Eustachian catheter.

Secondary nasal syphilis is a disease of infancy, taking the form of a persistent nasal catarrh which goes by the name of "snuffles," and is easily recognized.

The common form of nasal syphilis is the tertiary gumma—its favourite seat is the bony septum, and its development is characterized by nasal obstruction and pain, which may be severe. The process is often very destructive, not only causing a large septal perforation, but extending to the skin surface and, especially in hereditary syphilis, giving rise to great disfigurement.

Necrosis of bone is an important diagnostic feature, and black fetid sequestra are often exfoliated. This bone-necrosis at once differentiates the disease from ozæna, with which it might otherwise be confounded. From lupus it is distinguished by its more rapidly destructive effects and by the absence of the "apple-jelly" lupus nodules on the skin. Malignant disease is usually unilateral and begins after middle life, but it may, at times, resemble syphilis closely.

Syphilitic perforation of the nasal septum affects the bony part, thus differing from perforation due to other causes.

As a result of cicatricial contraction during healing, the patient may be left with the characteristic deformity known as "lorgnette nose."

SYPHILIS OF THE LARYNX.

Secondary syphilis may be accompanied by hoarseness, but laryngoscopy will merely reveal a laryngitis with no definite character.

Of greater frequency and importance are the laryngeal lesions which occur in the tertiary period. As has been noted, the term "tertiary" is rather indefinite, but may be held to include those affections which appear after the second year of the disease. Laryngeal syphilis may then take the form of a diffuse infiltration, involving the whole larynx, but often more noticeable in the arytenoid region. The syphilitic tendency to contraction is well marked, and, owing to its gradual development, may cause no dyspnœa until the air-passage has become greatly narrowed. Pain, as a rule, is slight, and the only symptom may be an obstinate and persistent hoarseness. Septic infection may lead to perichondritis of the epiglottis or arytenoids, resulting in the formation of a large abscess and sloughing of the cartilage.

Diagnosis, from the laryngoscopic appearance, is often a matter of considerable difficulty.

Gummatous ulceration is infrequent. The syphilitic ulcer has a sloughy, "wash leather," floor and sharply defined red edges. The absence of pain is a notable feature in syphilis of the larynx, in contradistinction to cancer and tubercle.

The former, as a rule, affects older persons and progresses more slowly than syphilis, but in many cases only continued observation and the therapeutic test (anti-syphilitic treatment) will decide the question. In tuberculosis of the larynx, the pain is often considerable, and the laryngeal lesion is always secondary to pulmonary tuberculosis.

SYPHILIS OF THE EAR.

External and Middle Ear.—The sound-conducting apparatus is rarely touched by syphilis. Chancre of the auricle, and also tertiary lesions, are very unusual.

Secondary eruptions on the skin may, naturally, affect the external ear, and condylomata may attack the meatus, sometimes causing intractable ulceration. Diagnosis presents no difficulty, as syphilitic lesions are always present in other parts.

Syphilis of the middle ear is probably not uncommon, but there is no certain means of ascertaining whether an aural suppuration is syphilitic or not. Catarrhal or suppurative otitis media may, of course, be encountered in syphilitic subjects.

Inner Ear.—It is against the inner ear and its associated nerve elements that syphilis wields most frequently its destructive influence. About 5 per cent. of syphilitics are affected in this region. It is a well-known fact that syphilis may attack the cranial nerves, optic, oculo-motor, facial, etc., alone or in combination, but the most vulnerable of them all is the auditory or eighth nerve. In 104 cases studied by Benario, the auditory nerve was affected in 51, the optic in 37, the facial in 9, and the oculo-motor in 8. The cochlear branch of the auditory nerve may alone be involved, or the vestibular branch alone, or both branches together. Syphilis of the vestibular apparatus give rise to the so-called Ménière symptoms, viz., nystagmus, vertigo, and ataxia, while lesions of the cochlear are, of course, characterized by loss of the hearing function. The sudden onset of bilateral and rapidly progressive deafness with diminution of bone conduction should lead one to suspect syphilis. Sometimes the development is gradual, and in such cases the deafness is often preceded by headache and tinnitus. Unless there is a coincident or previous otitis media, the tympanic membranes will appear normal. In the early stages the loss of hearing affects the high notes, but the lower end of the scale may also be involved, so that the lower tone-limit is heightened. A characteristic sign is the shortening of bone conduction, as revealed by the tuning fork. Thus, the Rinne test is positive, and Schwabach test (comparison of one's own bone conduction with that of the patient) is shortened. Inflation of the middle ear is followed by no improvement in hearing.

Syphilis of the inner ear may appear at any stage of the disease, although usually it is a late phenomenon. Politzer has reported a case in which the deafness set in on the seventh day after infection.

The frequent involvement of the auditory nerve in syphilis has led some authorities to suppose that many of those cases are, in reality, a toxic neuritis resulting from the use of salvarsan or other arsenical compounds. But the symptoms have appeared in cases treated with mercury alone or with salvarsan in minimum doses. Furthermore, this nerve-deafness has not been observed after the use of salvarsan for non-syphilitic diseases.

Hence, it is now recognized that the involvement of the auditory nerve in syphilis is directly due to the syphilitic virus, and is, indeed, a "neuro-recurrence" and not an arsenical neuritis.

In congenital syphilis, deafness may appear at about the age of puberty, and as the patients usually present other "stigmata," such as keratitis, notched teeth, and fissures of the lips, the diagnosis presents little difficulty. Females are more susceptible than males. Prompt and energetic treatment is indicated, both in acquired and hereditary syphilis of the inner ear, if the residual hearing is to be saved, but the prognosis is never favourable.

TREATMENT.

(a) *General Treatment.*—Although the present-day methods of treating syphilis are familiar to most practitioners, a few notes on the subject may not come amiss.

In syphilis of the throat, nose, and ear, local applications must, of course, be altogether subsidiary to general measures.

The best known remedies are—

1. Mercury.
2. Iodide of potassium.
3. Arsenical preparations.

1. *The Use of Mercury.*—Of the many modes of administering mercury, the oral method is the most convenient and the most widely adopted. Protoiodide of mercury is held in great repute by the French, but it would be difficult to improve upon the "small grey-powder pill" which Jonathan Hutchinson prescribed so largely, and which contains one grain each of grey powder and Dover's powder.

Though less active than injections, oral administration of mercury is so simple that it must remain the method of choice when the patient cannot be kept under constant supervision. It stands to reason that careful directions must be given regarding the hygiene of the mouth, if stomatitis is to be avoided. It is also necessary to keep a watchful eye on the urine, whether we are giving mercury or arsenic, so that the first traces of toxic albuminuria may be detected. Inunction is a disagreeable, though effective, method of giving mercury and, like calomel fumigation, is now mainly of historic interest.

The intramuscular injection of one or other of the insoluble preparations of mercury is a good and exact procedure, the injections being made once a week.

Grey oil is to be preferred to calomel cream, for the latter is too painful for general use.

Should one decide to employ soluble salts of mercury (perchloride, benzoate, biniodide, etc.), the injections must be made daily, and the effect is no better than that following the weekly use of grey oil.

A method of treatment, which was and, I understand, is still adopted by Brocq at the St. Louis Hospital in Paris, consists of the intravenous injection of cyanide of mercury. This salt of mercury is one of the most stable. It contains a high percentage of the metal (80 per cent.), it is readily soluble, and it does not coagulate albumen. A one per cent. sterile aqueous solution is employed, and 1 cc. of this is injected into any prominent vein by means of an ordinary hypodermic syringe.

The injection is painless, the drug is rapidly absorbed, and its action is prompt and effective. One possible disadvantage is the occasional occurrence of diarrhœa from the action of the mercury on the alimentary tract, but this only occurs after the initial dose, and may be prevented by giving the patient 5 grains of Dover's powder at the time of injection and by beginning treatment with a small dose (½ cc.) of the cyanide solution.

Injections are practised daily or every second day. In combination with novarsenobenzol, "914," as described below, I have found the intravenous use of cyanide of mercury a valuable remedy, when one is anxious to bring the patient rapidly under the influence of anti-spirochætal drugs.

2. *Iodide of Potassium.*—This drug in itself does not cure syphilis. Its chief value lies in its power of promoting the absorption of connective tissue, so that anti-syphilitic remedies may reach the lurking places of the spirochæte. Potassium iodide is, therefore, of greatest value during the tertiary stage, in the intervals between the courses of mercurial and arsenical treatment. Iodide has a further important action. Like a visit to a sulphur spa, it favours the elimination of mercury and is, consequently, of great value in old-standing syphilis. As a rule it is given

by the mouth, in dilute solution, the dose being 10 to 30 grains thrice daily.

To counteract its depressing effects, it may be combined with small doses of nux vomica, while belladonna is said to neutralize the toxic effects. A strict watch must be kept for these effects, known as "iodism," of which the most prominent symptoms are coryza, œdema of the eyelids, gastric derangement, and a rash resembling acne. From the laryngological point of view, the most important phenomenon of iodism is œdema of the larynx, which may even call for tracheotomy. In susceptible patients quite a small dose of iodide may cause laryngeal œdema.

Ricord had a case necessitating tracheotomy, after the admininstration of only 7 grains of iodide. The important practical point is that care should be exercised in the use of potassium iodide in syphilis of the larynx, by which affection the larynx is already predisposed to œdema.

Harrison advises the use of iodides in short intensive courses, rather than in small doses spread over a long period.

3. *Arsenical Treatment.*—Although arsenic had long been used in the treatment of syphilis, its value was not fully realized until the discovery of Ehrlich's "606," or salvarsan, now generally known as "arsenobenzol." At first it was hoped that a single dose of this substance would cure any case of syphilis, but this view was very soon proved to be fallacious.

To attain permanent results, the drug must be used just as persistently as mercury.

Although a potent remedy, arsenobenzol is not free from disadvantages, not the least of which is the complex technique of its preparation and administration.

In the newer substance of similar nature—"914," or neosalvarsan, now called novarsenobenzol—we have a compound which, although weaker in action than arsenobenzol, is easier to administer and is very efficacious. Neokharsivan and novarsenobillon are practically identical with novarsenobenzol.

Unlike its predecessor, novarsenobenzol need not be neutralized with carbonate of soda, but is merely dissolved in water prior to use. Simplest of all is the method of Ravaut, who showed that a concentrated solution could be injected and that the average dose (·45 grain) might be given in 10 cc. of cold water.

Ravaut has lately advised the use of solutions even more concentrated. Distilled water, boiled and cooled, is employed, the technique of the intravenous injection being readily acquired with a little practice.

Systematic treatment must be insisted upon in every case, the nature of the disease and necessity for long-continued treatment being carefully explained to the patient at the start.

The Wassermann Reaction.—This test has been widely adopted as a means of diagnosis and as a controlling factor in treatment, but too great stress should not be laid upon the results. Of prime importance is the fact that a negative result is of no value. Negative Wassermann need not imply the absence of syphilis. It is also well to remember that the test is valueless in the early stages of primary syphilis. The largest percentage of positive results is obtained during the secondary period, while in the tertiary stage only about 70 per cent. of the cases react, even if active lesions are present.

Indeed, it is significant that Thibierge, in his recent work on *Syphilis et l'Armée*, states that he has ceased to employ the Wassermann test, so varying and discordant are the results.

Of other arsenical compounds, such as hectine, galyl, etc., I have little personal experience. Galyl is a favourite with many, though it is a weak preparation and somewhat varying and uncertain in action.

The most recent innovation, a complex substance derived from antimony, known as disodoluargol, is stated to be more powerful and efficient than arsenobenzol, but has hardly yet passed its experimental trials.

From what has been said, it will be seen that each case must be judged separately and treated differently. There is no definite routine treatment for syphilis, though every authority has his own favourite method.

(b) *Local Treatment.*—Although altogether subsidiary to general medication, local applications will frequently contribute to the patient's comfort and recovery. It is hardly necessary to mention the importance of the care of the teeth, and the avoidance of alcohol and tobacco, in cases of bucco-pharyngeal syphilis.

1. *The Pharynx.*—In primary tonsillar syphilis, enucleation of the tonsil is of as little value as is the excision of chancres elsewhere. The application to the sore of equal parts of pure carbolic acid and tincture of iodine is a good line of treatment.

Mucous patches may be cauterized with nitrate of silver, after cleansing with peroxide of hydrogen, A more powerful caustic is acid nitrate of mercury, which must be applied in small quantity and with great caution. In view of the smart reaction which follows cauterization, this treatment should not be practised too frequently or over too great a surface. For secondary lesions, as indeed for all stages of buccal syphilis, a simple alkaline mouth wash is acceptable to the patient. Another favourite prescription contains equal parts of lotio nigra and saturated aqueous solution of chlorate of potash.

For the gummatous ulcer, painting with chromic acid solution (10 grs. to the ounce) is a useful treatment, whilst tincture of iodine had the approval of Fournier. Large tertiary ulcers of the tonsil are best dressed with an insufflation of powdered calomel. Pain is relieved by powdering with orthoform, alone or combined with iodoform or resorcin. The treatment of naso-pharyngeal stenoses is essentially surgical, but is seldom very satisfactory. It is well to be certain that all trace of active disease is gone, before any operation is attempted.

2. *The Nose.*—Syphilitic coryza in infants is locally treated by cleansing the nostrils with peroxide or an alkaline lotion, followed by the installation of a few drops of menthol in olive oil (1 in 100 solution).

The mutilating effects of tertiary nasal syphilis demand that this phase of the disease should receive intensive treatment. The nose should be irrigated daily with warm alkaline lotion, to which a little carbolic or lysol has been added. After the diseased area has been cleansed and purified in this way, the dressing should be completed by the insufflation of powdered calomel. A useful prescription contains 1 part of calomel and 10 parts of bismuth subnitrate. Mandl's paint (iodine, potassium iodide, and glycerine), so valuable in ozæma, may also be applied to syphilitic cases. When sequestra are present, antiseptic applications must be given a prominent place. As soon as the bone has become loose, its expulsion may be aided with forceps, after applying cocaine, but such intervention should not be too vigorous, lest severe hæmorrhage be the result. The mass may have to be broken up *in situ* before it can be removed.

3. *The Larynx.*—Affected surfaces should be sprayed with peroxide and then powdered with diluted calomel

If iodide is being used in general treatment, a careful outlook must be kept for the possible laryngeal œdema. Cicatricial stenosis of the larynx often develops insidiously, and tracheotomy may at any time become necessary. The prognosis of such a condition is seldom favourable, cicatricial stenosis being one of the most unsatisfactory of all laryngeal affections, so that a tracheotomy tube may have to be worn permanently. Energetic general treatment may, in some instances, result in a re-establishment of the airway.

4. *The Ear.*—The unfavourable outlook in syphilis of the inner ear can readily be understood when it is remembered that this lesion is simply an expression of the most serious type of syphilis—syphilis of the nervous system. If lumbar puncture is performed in such cases, the fluid will be found to contain an excess of lymphocytes and to give a positive Wassermann reaction.

Local treatment has no place in such a malady, and even energetic general treatment may be of little value. In treating these deep-seated forms of syphilis, the initial dose of arsenobenzol, or of its allies, should be small, on account of the reaction which may result.

The intra-spinal injection of salvarsanized serum is said to give better results than intravenous injections.

This method, introduced by Swift and Ellis, consists in administering a dose of arsenobenzol, and, an hour later, drawing off some blood from the patient. The blood is centrifugalized, and the serum injected by lumbar puncture.

Addisonian (Pernicious) Anæmia at an Advanced Age.

WITH SOME NOTES ON THE DIAGNOSIS OF THIS DISEASE.

By GORDON WARD, M.D.

THE patient, Mrs. H. B., aged 85, came of a long-lived family, for one sister had lived to the age of 86 and another to 96. She herself had never enjoyed robust health, but had been no more frail than usual until three or four years before I saw her. She had then noticed, coincident with the heavy labour of nursing a helpless husband, progressively increasing weakness. At the same time, she began to suffer from attacks of nausea and flatulence. Somewhat later her friends noticed her increasing pallor, and she began to suffer from a glossitis which was thereafter almost the most prominent feature of her illness. Not long before I first saw her, œdema of the legs was noted, and the gastro-intestinal symptoms had assumed the form of severe recurring crises, accompanied by nausea or vomiting, very considerable spasmodic pain and, rarely, diarrhœa. She was treated as a case of malignant disease—a diagnosis which is frequent in this particular type of Addisonian anæmia.

When I first saw her, which was six months before her death, she presented the following characteristic symptoms, which permitted a very confident diagnosis of Addisonian anæmia before the blood was examined. It may be remarked that in a straightforward case, this should almost always be possible.

Firstly, she was very pale and had a yellowish colour. In so old a lady this had not previously called for comment, and was only noticed by myself when the character of the glossitis had aroused my suspicion. With this pallor was a constant complaint of weakness. There was further œdema of the ankles. One therefore felt entitled to assume that the pallor was not that of age, but was due to a severe anæmia.

Secondly, she had certain gastro-intestinal symptoms with glossitis. The former were not continuous but came in paroxysms. Rarely these were excited by particular foods, on one occasion, tomatoes. During a paroxysm she felt "knots" in her stomach, and these were apparently due to localized constrictions, spasms, of the bowel. Added to these there was a continual sense of soreness in the abdomen. The tongue was small and pale, with little suggestion of the ordinary markings. It presented several red spots of various sizes, which the patient referred to as "ulcers," and which were in fact of this nature. They were very painful.

Thirdly, she complained of numbness of the extremities. No very great stress could be laid on this at her age, but it was all that was detected of the nervous symptoms of Addisonian anæmia. No doubt two-point discrimination tests and other refinements might have brought further minor abnormalities to light, but these were not considered to be necessary in the circumstances.

Supposing that these three classes of symptoms were related in the one morbid process, there could be no question of the diagnosis. Severe anæmia does not produce glossitis, hence they were dependent on a common cause, or the glossitis had produced the anæmia. However that may be, there are only two diseases in which glossitis and severe anæmia commonly co-exist. These are tropical sprue and Addisonian anæmia The former was out of the question. Moreover, the other symptoms, particularly the gastro-intestinal spasms and the intense weakness, agreed with this diagnosis. The weakness in Addisonian anæmia is commonly out of proportion to the anæmia, and is probably in part dependent on the toxic factor

There are three means by which the diagnosis may be confirmed. The first is by determining the absence of hydrochloric acid from the stomach. If this occurs during a remission of the disease, it is an important aid to diagnosis; it is less important when the anæmia is severe, because severe anæmias from other causes, *e.g*, cancer, may also show this The second is by blood examination This was done, and the red cells were found to be two million, with the characteristic changes of shape, etc. The hæmoglobin was 48 per cent.—a percentage not incompatible with considerable activity in some people The third is the detection of urobilin in the urine. By the spectroscope this was found in considerable excess.

Having established the diagnosis, one had next to face the question of treatment. There was no prospect of cure, for there is still no cure for this condition, although long remissions may be observed. But arsenic has certainly a good effect in many cases and must be tried. I commenced with five minims of the Liq. Arsenical Hydrochlor. three times a day, together with an equal amount of the Liq. Morph. Hydrochlor. It is frequently difficult to get patients to take arsenic, because it produces pain, vomiting, etc. For this reason I add some morphia. Even then difficulties arise, and in this case arsenic had finally to be abandoned, which one would have been very loth to do in anyone who still felt that life was worth living, which was not the case with my patient.

The patient's chief complaints were of sore tongue and pain in the abdomen. For the former I gave 1 per cent of cocaine in glycerin, and later increased it to 2 per cent. The latter kept the soreness of the tongue completely in abeyance, but the feeling of numbness to which it gave rise was not much liked. However, the patient preferred numbness with ability to take food to soreness without such ability, and this prescription is one I have come to regard as most valuable.

For the gastro-intestinal symptoms I ordered such drugs as were necessary to regulate the bowels, viz., liquid paraffin and an occasional dose of senna pods. Thereafter gastro-intestinal symptoms gave no trouble as long as this *régime* was followed, except on the one occasion on which she took tomatoes. One must exclude a certain feeling of oppression, not soreness, which was not quite banished. There was no more vomiting.

With these measures the patient's red cells rose in two months to four million and a half, and the hæmoglobin to 96 per cent. She was able to go out into the garden and walk about the house. One month later she refused to take any more arsenic, as she said it made her sick. She had allowed herself to become constipated, which was probably the cause of the sickness. She was a somewhat masterful old lady, and as she was well aware of the value of arsenic in her complaint, her decision was not opposed. A month later she was distinctly not so well. Œdema of the feet had recurred, and her "ulcers" had returned to the tongue. She had by this time broken almost every rule of life prescribed for her. In a further two weeks she was back in bed with a badly ulcerated tongue. From this time on she complained also of pain in her throat on swallowing, and I suspect that she had ulceration of the œsophagus. She certainly had small ulcers on the inner and outer aspect of the lower lip, on the frenum and the gums. She grew rapidly but spasmodically worse, and died six months after I first saw her. Throughout her illness, so far as I saw it, constipation was associated with exacerbation of the disease

From a consideration of this case and many others in which similar points have arisen, one would like to emphasize strongly two points, viz. :—

(1) That malignant disease of the stomach without palpable tumour should not be diagnosed unless Addisonian anæmia has been excluded.

(2) That chronic or acute glossitis with anæmia is practically always Addisonian anæmia.

I can find only one case in the literature which in any way approaches the age of this patient. Laache records a case in a man of 73. No cause for the anæmia could be found. Schauman recorded a case of bothriocephalus anæmia at 73, but this would not now be considered to come within the Addisonian group. American observers report two cases between 70 and 80 from the literature; probably these are the cases already mentioned.

My case was therefore 13 years older at her death, which took place in her 86th year, than any other recorded case, while the onset of her disease was apparently about four years earlier.

A few years ago I saw with Dr. Fenouhlet of Herne Bay, a lady of 76 in whom the illness began with a swelling of the tongue so acute and so great that the tongue was hardly contained in the mouth. Gastro-intestinal symptoms, numbness and tenderness of the legs, and a blood-count of a little over one million reds completed the picture. The course of the disease was paroxysmal, and when I last heard she was somewhat improved.

Were it not for the first case, this one would have established a record for age.

I append details of the blood counts in these two cases:—

	Mrs. B. 11/6/19.	Mrs. B. 23/8/19	Miss R. 10/8/16.
Red cells - -	2,030,000	4,640,000	1,070,000
Nucleated reds -	None	None	38 per cmm.
Megalocytes -	The majority	Very many	Very many
Microcytes - -	Many	Present	Very many
Poikilocytes -	Many	Few and small	Practically all
Polychromasia -	—	Marked	Very marked
Stippling - -	—	?	Very marked
Hæmoglobin % -	48	96	33
Colour index -	1·2	1·04	1·605
White cells -	2,200	—	6,400
Polymorphs % -	41·0	44·8	45·2
Eosinophils -	2·0	6·6	3·8
Mast cells - -	0·5	2·8	0·4
Transitionals -	0·5	2·8	3·6
Small monos -	55·0	42·2	45·4
Large monos -	1·0	0·8	1·2
Myelocytes -	None	None	0·4

Ionization.

BY MARK WARDLE, L.R.C.P. AND S., V.D.

Hon. Surgeon, Bishop Auckland Cottage Hospital, etc.

HAVING worked at this method of treatment throughout the last year, I have found two outstanding results: (*a*) its wide range of usefulness; (*b*) its certainty.

In treating cases with ions, some important points must be kept in mind, viz.:

Correct polarity;

Accurate contact of the electrodes;

Protection of any tender surface by coating with a non-conductor (collodion, for instance).

In treating an area one need not worry about having contact with *every* portion of it, seeing that ions travel quite a considerable distance beyond the point of contact; for instance, the whole of the surfaces of such a large joint as the knee can be freely "douched" with ions driven from a pad placed over the knee—but much care must be exercised in the protection of delicate parts of the skin, or there will result burns that will take something like two months to heal.

I append notes of some cases, chosen as being the worst of their particular kind, and on account of their diversity of character.

1. *Chronic Gouty Arthritis of Knee.*—The slightest movement caused severe pain. X-ray photo showed osteophytes at several points, and considerable denudation of patellar cartilage. *Treatment.*—Two per cent. solution of potass. iodid. in pad placed over knee. 50 M.A., 30 min., alternate days. After first application pain almost gone. After second, patient walked for an hour in comfort. Regular application for four weeks, occasional afterwards. X-ray photo three months after: no osteophytes visible; use of limb normal.

2. *Chronic Ulcerative Keratitis.*—Entire eye-ball inflamed; sight

almost nil. Several months' treatment by numerous methods resulted in no improvement, and excision to save the good eye seemed the only thing left. Zinc ions by pad over closed eye. 5 M.A., 10 min. alternate days. After first application improvement commenced. After seventh, ulcers all healed; no opacity, the rest of eye-ball free from inflammation and sight normal.

3. *Polypoid Growth*, with extensive granulations involving anterior portion of tympanic membrane and adjacent parts, free discharge of fœtid pus; could hear only sharp loud sounds. Zinc ions, 8 M.A., 10 min. alternate days. Symptoms steadily improved; treatment continued for 20 applications (seven weeks). All growths gone; no discharge. Three months later no return of symptoms; can hear watch.

4. *Chronic Rhinitis.*—Much fœtor and discharge. Zinc ions, 10 M.A., 10 min. alternate days. Continued for 20 applications. Two months later condition normal.

5. *Spinal Curvature Dorso-lumbar.*—Of many years' standing (tuberculous?). Patient could only move about room by holding to furniture, and suffered much pain. Iodine ions (sol. potass. iodid.), 50 M.A., 30 min. alternate days. Pain relieved after first application; gradually disappeared. After twentieth, walks without help and gets up and down stairs.

6. *Carbuncle.*—Had reached stage of frequent attacks of severe pain; slough firmly adherent. Zinc ions, 15 M.A., 30 min. One application; pain ceased during application. Twelve hours later slough came away adhering to dressing.

7. *Ophthalmia Neonatorum.*—Baby fourteen days old. Lids of both eyes bulging and inflamed, pus welling out. Zinc ions, 3 M.A., 3 min. After first application, eyelids normal; slight amount of pus. After second application, left eye normal; slight indication of pus in right, child can open its eyes freely. No sign of corneal injury.

8. *Sinus after Mammary Abscess (Tubercle)*.—Foul discharge. No improvement under various treatment. Running irregular, temperature up to 102. General and pulmonary condition getting steadily worse. Sinus slit up. Zinc ions, 20 M.A., 30 min. After first application discharge slight. No fœtor. Subsequent improvement steady; temperature normal. Second application given a fortnight later. General and pulmonary condition, a month later much improved.

9. *Inoperable Cancer of Rectum.*—I have already reported this case in the *B.M.J.* (October 18). There is now (November 20) only a slight band of malignant growth at the anus, and I am confidently awaiting the termination of the case to prove that we have in zinc ions an efficient agent for the "cure of cancer."

Ruptured Popliteal Aneurism.

By CECIL P. LANKESTER, M.R.C.S.
Assistant Surgeon, Royal Surrey County Hospital.

A MAN, aged 49, was admitted under my care on March 23, 1917, with the history of having been kicked by a camel on the left knee in 1903, and having had recurrent attacks of synovitis in the same knee about every 12 months. He had strained his knee a month before admission. There was no history of syphilis Three weeks before admission there was a swelling in the left leg above the knee, which had increased especially during the last week: also he complained of pain in the thigh and spine.

On admission, his arteries were somewhat hard, there was a large pulsating tumour on the inner side of the left thigh, just above the level of the patella, no apparent attachment to bone, pulsation could be seen, and the tumour was tense. A loud to-and-fro bruit could be heard over it.

During the week after admission the tumour increased in size, and believing it to be a popliteal aneurism, I arranged to operate on April 1, hoping to have the help of a consulting surgeon whom I expected on that day.

Early on the morning of April 1 the patient had a sudden attack of pain and felt "something burst." Examined on the table ready for operation on that afternoon, when I first heard of this attack, the tumour appeared harder, less defined and more tense, it had ceased to pulsate, and no murmur could be heard. The consultant having examined it expressed the opinion that the most probable diagnosis was one of sarcoma, and advised amputation, which I arranged to perform on April 5.

On the 3rd there was slight pulsation on the internal posterior surface of the tumour, and a faint bruit was audible at the upper internal surface near the front, also on the external surface of the limb.

Before proceeding to amputate on the 5th instant, I thought the patient should have the benefit of the doubt and made an incision over the tumour and found a quantity of extravasated blood and a burst aneurysmal sac immediately below the passage of the femoral artery through the adductor magnus muscle, in size about that of a hen's egg, the entry and exit of the artery could easily be seen. The artery was tied above and below, and a short tube inserted (this was removed next day without disturbing the main dressing).

The patient made an uninterrupted recovery, and was discharged on May 19; 18 months after the operation, he reported himself perfectly well.

Cerebro-Spinal Fever:

RELAPSE TEN WEEKS AFTER RECOVERY FROM ORIGINAL ATTACK.

By PHILIP N. RANDALL, M.B.
Medical Superintendent, Bromley and Beckenham Joint Isolation Hospital.

I case here reported may be found of interest, because Sir Thomas Horder tells me that true relapses—using the term in its proper sense to exclude recrudescences or exacerbations, a common feature in many cases—are distinctly uncommon in this disease. The term, he adds, should not be used until and unless all the signs have subsided (that is to say, absence of Kernig's sign and a normal pulse-rate), because the very important point is involved whether a relapse is due to reinfection from the throat.

A man, 33 years of age, was admitted to the Isolation Hospital, Bromley, on November 18, 1919, for cerebro-spinal fever, with the history that he had suffered from an attack of this disease at Eastbourne in August, 1919, and had been discharged well from hospital towards the end of September. He was advised at the time not to undertake any work for a few months, and returned home to Beckenham. In November he was offered light work on the roads, sweeping up leaves; as he felt quite well, and was anxious to accept the offer, he was allowed to do so. After getting wet through whilst at work, he was, on November 13, seized with pain in the loins which rendered him incapable for work. On November 16, this lumbar pain had become so severe that he had to stop in bed, and arrangements were begun to get him admitted to St. Thomas's Hospital. On November 17 vomiting came on, and on the morning of the 18th, as he was reported to have intense headache and severe pain in the neck, his immediate admission to the Isolation Hospital was recommended. On arrival there, it was found that any attempt to flex the head on the chest gave rise to very acute pain in the neck, where the muscles were rigid and very tender on pressure; there was, too, slight retraction of the head. Kernig's sign was present, and the attempt to extend the leg upon the thigh caused extreme pain in the lumbar region.

He was conscious, but had lost control of himself owing to the intense headache. Lumbar puncture was performed under chloroform, and 20 cc. of semi-viscid, thick, purulent-looking fluid, which flowed very slowly out of the needle, were obtained. An injection of 25 cc. of anti-meningococcic serum (mixed types) was given. This brought about some relief for a few hours, but the headache and pain then returned in their original severity.

Films from the cerebro-spinal fluid showed pus-cells in great abundance, and the presence, intra and extra-cellular, of the meningococcus in large numbers. A culture of this organism was obtained from the fluid on nasgar.

On November 19, 50 cc. of fluid, a little less viscid in character, were obtained, and serum was injected. Lumbar puncture was performed each day subsequently, followed by an injection of serum. The fluid on each occasion was thicker, more viscid, and more difficult to obtain. Only 20 cc. flowed out on the 20th, and 10 cc. on the 21st and 22nd. The fluid on the 23rd was much too viscid to flow out at all, and an attempt to draw it out by suction yielded less than 5 cc. The intense headache continued throughout, but was controlled to some extent by occasional injections of morphia. The temperature was normal on admission and remained at about that level throughout, rising suddenly to 100° on the 19th and to 102° on the 20th, falling quickly again on each occasion. The pulse-rate was most irregular, varying between 64 and 116. Death ensued during the evening of November 23.

Dr. Willoughby, M.O.H. of Eastbourne, has very kindly supplied details of the original attack in August. The patient was admitted to the Isolation Hospital on August 14, being the tenth day of his illness, when Dr. Willoughby had been called into consultation and considered the case to be one of cerebro-spinal fever. Lumbar puncture was performed seven times without an anæsthetic, and five injections of 30 cc. of serum were made. Iodide of potash, hexamine, and an occasional injection of morphia, were given as well. There was little or no real delirium. The diplococcus was present in the cerebro-spinal fluid. The attack took a straightforward course, and the patient was discharged from hospital on September 23.

I am indebted to Dr. P. J. Curtis, of Beckenham, for the history immediately antecedent to the patient's admission to hospital in November.

Practical Notes.

Migraine and Anaphylaxis.

In fairly numerous instances, migraine seems to yield to a very simple treatment. This consists in giving, three-quarters of an hour to an hour before the three chief meals, a cachet containing 0·50 g. of peptones of good quality. The treatment does not aim at the attack itself, but rather at modifying the general condition, unknown as yet, to which the return and the severity of the attacks are due. Attacks of migraine get less during the period of treatment by peptones, and may disappear altogether at the same time as the digestive troubles affecting the patients are remedied. All these patients, in fact, are more or less dyspeptic.

Pagniez, Pasteur-Valléry-Radot, and Nast have, in addition, observed a very intense urticaria set up by all albumens, animal or vegetable, and the anaphylaxis in respect to these appears to be absolute. Relief was first obtained by taking a very small meal before the large one, this was greatly simplified by giving a cachet of peptone before the meal, and has proved successful in those suffering from migraine. It is the method of Besredka applied to alimentary anaphylaxis.

This anaphylactic theory of migraine seems to be supported by the good results obtained by treatment with peptones. Laumonier, however, whilst admitting the part played by anaphylaxis in urticaria, considers it doubtful in the case of migraine

Whatever may be the explanation of the eupeptic or anti-anaphylactic effect of peptones, the results obtained in migraine are important and interesting.—(*Journ de Méd. et de Chir. prat*, October 10, 1919.)

Intra-pleural Injections of Methylene-blue for Empyema.

Intra-pleural injections of methylene-blue were recommended by Nobécourt in 1915, and seemed to be of very great use, especially for cases in which the patients were too infected and too much weakened to be able to bear pleurotomy. Charier reports seven out of eight children treated in this way were cured successfully.

The solution used consists of 5 g. of methylene-blue in 95 g. of water sterilized by prolonged boiling. Solution takes place slowly. After standing for 12 hours, the solution is filtered through a sterilized paper, washed with boiling water, and the solution is collected in a sterilized flask.

After drawing off the fluid from the chest, 10 cc. of the solution described, slightly warmed, are injected into the pleural cavity, the injection being made very slowly if the quantity of purulent fluid withdrawn has not been very much.

The injection is often followed by profuse sweating, relief of the dyspnœa, and a fall in temperature. The last, however, may fail to occur.

If the amount of fluid withdrawn has not been large, the patient sometimes experiences, some few minutes after the injection, very great pain in the side and a passing feeling of oppression, but it is very seldom that an injection of morphia is required to relieve this.

The elimination of the methylene-blue by the kidneys begins in the third hour after the injection, but the pleural fluid will be found to be still coloured in a fortnight or three weeks later. Nobécourt has, on rare occasions, observed blue sputa.

In very serious cases, this treatment will not be enough to bring about a cure, but it is of very great use in restoring the patient's strength, improving his infected condition, and thus bringing him into better condition for pleurotomy.

Professor Hutinel uses this method systematically for all cases of purulent pleurisies in his practice at the Hôpital des Enfants. Charier's patients were from five to fourteen years old. Nobécourt had good results from its use, in the same dose, for soldiers, much weakened by purulent pleurisy after measles. These attacks, for the most part, had been preceded by severe broncho-pneumonia.

In infants, suffering from pneumococcal empyema, Nobécourt and Parat prefer to use intra-pleural, or even intra-pulmonary, injections of 10 cc. of anti-pneumococcic serum.

In adults, Emile Weill and Loiseleur have obtained good results by injecting 2 cc. of the 5 per cent. solution, but they inject air after tapping the chest so as to ensure that the pus is completely evacuated. The methylene-blue is only injected then.—(*Journ. de Méd. et de Chir. prat.*, October 10, 1919.)

Treatment of "Carriers" of Diphtheria.

Professor Arloing, of Lyons, points out that the serum-treatment of diphtheria has been made more effective by the introduction of a serum which is both antitoxic and bactericidal for the diphtheria bacillus. It is obtained by injecting into the horse the diphtheria toxin and the bacilli together. By use of this the bacilli present in the pharynx and on the tonsils can be reached by making the patient swallow tablets prepared with this serum, or better still by insufflation of the throat with dried serum, well powdered, mixed with an inert powder and with gum-tragacanth, in order to increase its adhesion to the mucous membrane and thus ensuring a longer contact with the bacilli to be destroyed. The insufflations are made three or four times a day, between meals, after the patient has gargled with some antiseptic lotion. In three weeks, very often in less, the diphtheria bacilli will be found to have disappeared from the throat. The procedure is quite simple and causes no pain. Two bacteriological examinations of the throat should be made after giving the insufflations, the second to be taken a week after the first.—(*La Médecine* December, 1919.)

Reviews of Books.

The Physiology of Muscular Exercise. By F. A. Bainbridge, M.D., F.R.S. (Monographs of Physiology). Pp. x + 215. London: Longmans, Green & Co. 10s. 6d. net.

On the principles underlying the behaviour of the body during muscular exercise to a large extent depend the treatment of the unfit, the building up of the sound body, and the making of an "A 1" population. The author of this monograph points out that muscular exercise presents three problems. The first is the changes taking place in the skeletal muscles, whereby the transformation of energy, which constitutes muscular movement, is effected; the second is the nature of the adjustments occurring elsewhere in the body in order to provide the muscles with the oxygen and foodstuffs required for this purpose, and the third is the means by which these are interwoven and bound together to produce the fabric of muscular exercise. The second and third of these questions are mainly dealt with in the pages of this book, the first one being considered only so far as it bears upon the general argument. In successive chapters we find an adequate discussion of the sources of muscular energy, the respiratory changes during exercise, the effects of exercise upon the heart and the manner in which it responds, the consumption of oxygen, training and its effects, over-stress, and exercise at high altitudes The matter is put together in a very readable form and the practitioner will find much of interest to him in the treatment of disabilities and disease. A useful bibliography completes the book.

Essentials of Physiology. By F. A. Bainbridge, M.D., F.R.S., and J. Ackworth Menzies, M.A., M.D. Pp. viii + 484. Third edition. London: Longmans, Green & Co. 12s. 6d. net.

This manual of physiology gives in concise form the fundamental facts and principles of physiology, such as are required by the pass student, space being gained by the exclusion, so far as possible, of histological details and descriptions of chemical and experimental methods.

In this third edition, the changes are trivial and are principally confined to the sections dealing with the constitution of the proteins, the chemical changes accompanying muscular contraction, and the functions of the renal tubules, which have been re-written; but the whole text has been revised and brought up to date.

Metabolism is well treated, and a section is devoted to accessory food factors. The ductless glands and their functions are clearly discussed, and the work of Paton on the function of the parathyroids in guanidin metabolism is noticed. The electrical phenomena of the heart as detected by the string galvanometer are described. A lucid account is given of the structure and functions of the various parts of the nervous system. Altogether, the book is one that can be confidently recommended as giving an excellent account of modern physiology, and it is freely and adequately illustrated.

Organic Chemistry for Students of Medicine. By JAMES WALKER, LL.D., F.R.S., Professor of Chemistry, University of Edinburgh. Pp. xii + 322. Second edition. London: Gurney and Jackson. 10s. 6d. net.

PROFESSOR WALKER has written this book primarily for the medical student, and in order to deal with the subject of organic chemistry has selected those chemical substances as illustrations not so much for their importance in systematic or synthetic chemistry as for their medical interest. In this way time and labour are saved to the student without, Professor Walker maintains, any diminution in the appreciation of scientific method. We believe this to be quite a sound principle and one which might be extended to other subjects of the preliminary scientific curriculum, *e.g.*, biology and physics.

After a preliminary summary on the methods of organic chemistry, methane and its derivatives are considered, thus giving an opportunity for a full description of formaldehyde, chloroform, and iodoform. Then follows a section on the carbohydrates (they are dealt with at length later) and fermentation, leading on to ethyl alcohol and ethyl chloride. In this manner the whole of the essentials of organic chemistry are considered, including the proteins, lipoids, alcohols and dyes, stains, and indicators. Much more space is devoted to the alkaloids than is usually found in books on organic chemistry and the organic arsenic compounds, atoxyl, cacodylic acid, and salvarsan, are fully considered.

We consider this book to be well suited to the requirements of the medical student, it is clearly and interestingly written and well printed and produced.

of Venereal Diseases. By A. C. MAGIAN, M.D. Pp. 215, with 61 illustrations. London: William Heinemann, Ltd.

WE have no hesitation in saying that this is the best small book on venereal diseases we have had the pleasure of reviewing. As we anticipate a second edition will shortly be called for, we would suggest to the author to omit the multitude of remedies mentioned under treatment, limiting himself to those he has found most useful. Practitioners require information from the author's experience and prefer a certain amount of dogmatism to a concatenation of the sayings and doings of others. In our opinion all books should have the personal touch about them, as culling from others so often leads to the appearance of misstatements. The illustrations might be better selected as, in the first place, practically no instruments are required for the proper treatment of venereal diseases; and, in the second place, the beginner could learn more from a few photographs of chancres and rashes. The number of figures depicting Frenkel's exercises make the book unevenly balanced.

The Venereal Problem. By E. T. Burke, M.B., B.Ch. Pp. 208, with 6 illustrations. London: Henry Kimpton. 7s. 6d. net.

Although the author gives a brief description of the venereal diseases and a shorter one still of their history, the book is really intended to point out their prevalence in Great Britain; their effect upon the national life, and the factors causing their propagation and spread. It is a book which should be read because the author has very definite ideas concerning methods for the control and eradication of venereal diseases. In view of the vacillating standpoint taken up by many and the separation of medical opinion into two camps, a fact which makes the "man in the street" wonder whether we know anything about the subject, we would suggest to the author to write a book on the ethical side in more detail, to leave out the clinical and pathological sides, as such a book must necessarily lead to the cramping of the latter. The illustrations would also be better omitted as being too crude

Handbook on Skin Diseases. By Frederick Gardiner, M.D., B.Sc. (Public Health), F.R.C.S.E. Pp. 158. Edinburgh: E. and S. Livingstone.

In publishing a small handbook on skin diseases which is suitable for students Dr Gardiner has filled a long-felt want, especially in England where a systematic course in dermatology is not a compulsory part of the medical curriculum and where, in consequence, a small book to supplement the knowledge gained by the students in their casual visits to the skin departments of their hospitals should be of considerable value. Dr. Gardiner's *Handbook on Skin Diseases* seems eminently to meet this need; it is concise and yet contains considerable information, it is set forth in clear language, and it is illustrated by a series of photographs from the author's collection the majority of which are sufficiently characteristic for the diseases they portray to be easily recognisable.

and the Principles of their Evaluation. By Llewellyn J. Llewellyn, M.B., and A. Bassett Jones, M.B., with a section on "Pensions in Relation to the Eye," by W. M. Beaumont, M.R.C.S. Pp. 702. London: William Heinemann (Medical Books), Ltd. 30s net.

This is a large book, in which the authors have attempted the exceedingly difficult task of setting forth the principles which should govern the evaluation of disabilities or infirmities resulting from military service. Their laborious and painstaking work contains many hints and much information which is of value but, while fully recognizing this, it is impossible to feel that the authors have satisfactorily achieved the task they set out to accomplish. The main reason for this is the fact that no consistent principles have been observed in this country in establishing the pension system. Accordingly, an immense volume of regulations and decisions at present govern the administration of pensions, and these are continually changing, growing or being rescinded. The authors devote a large part of their volume to setting out the present rules, regulations, tables of assessment, etc., but already many of these have been changed since the volume was published, and we fear that in a very short time this large section of the book will be seriously out of date. In the second half of their book, the authors examine in detail the various considerations which should guide a Board in assessing the disability resulting from definite injuries. For major injuries, such as the loss of an eye or limb, or part of a limb, the assessment is already fixed in the table laid down by the Ministry of Pensions, but for injuries and mutilations not exactly covered by the official table this part of the book contains useful hints for arriving at the actual degree of disability. In many cases, the authors quote the rates of assessment accepted by various foreign countries for specific injuries, and this should be a further guide. The authors have included a brief and interesting account of the history of pensions from the earliest times.

National Health: from Magic, Mystery, and Medicine to a National Health Service. By FERDINAND REES, M.D. Pp. 68. Bristol: John Wright and Sons, Ltd. 1s. 6d. net.

THE author briefly reviews the craft of medicine as it was and as it is at present. He then brings forward temperate arguments in favour of a state service. He sees very clearly—*rem acu tetigit*—that the real obstruction to placing the medical profession in its rightful place as a profession is the fact that so many men engaged in its practice are wedded to the commercial side of their work. Unhappily, there are far too many in the profession who have mistaken their vocation, and should really be dispensing chemists. One recalls with a shudder the squalid wranglings in this connection that took place when the Insurance Bill was under discussion. It is manifestly hopeless to make any successful attempt to educate the laity on the subject of the actual uses and limitations of drugs, so long as these potion-mongers pander to the ignorant clamour of the public for " a bottle of medicine " wherewith to cure every ill to which human flesh is heir. It is most sincerely to be hoped, if the credit of medicine as a profession is to be set high, that those now leaving the schools may be filled with conviction of the higher ideals, and have the moral courage to carry them into practice. They will do well to take this brochure to heart, at any rate on this point.

Preparations, Inventions, Etc.

THE "FLUSOL" RADIATOR.

(London: The Brella Manufacturing Co., Ltd., 6, Dyers Buildings, E.C. 1.)

This is an apparatus for deodorizing, by which the disinfectant is slowly and continuously discharged from the container into the surrounding air through the meshes of the container.

In the container is placed a vessel for containing the disinfectant liquid used, a volatile oil or an emulsion. Two tubes pass in through the mouth of this vessel, in one of which is a wick which extends from the bottom of the vessel to within a few inches of the top of the perforated container. It is there bent back and passes to the bottom of the container outside the oil-vessel. The second tube is for refilling purposes, and extends to outside the container.

The space inside the container not occupied by the vessel and the wick is filled with an antiseptic absorbent substance, such as fine coke, absorbent fibre, sandalwood; sawdust, or previously treated wood-chips

The contents of the oil-vessel are slowly discharged by the wick into this packing and volatilized on the large surface there presented, the vapour passing out through the meshes into the air.

The fluid used in the radiator consists in the following proportions of—

Thymol	1	oz
Eucalyptus	6	,,
Turpentine	6	,,
Pine oil	½	,,
Wintergreen oil	½	,,
Ammonia (·880)	6	,,
Phenol	3	,,
Formaldehyde	4	,,
Cinnamon	3	,,

Water to 2 gallons.

The oil-receptacle holds 8 oz. If coke is used for the packing ½ oz. of naphthaline is added to it.

The radiator is a compact, neat arrangement which can stand on a table or be hung from the wall, and it appears admirably to carry out the purpose for which it is intended. The fluid needs renewing at fairly frequent intervals.

THE PRACTITIONER

MARCH
1920

Post-Dysenteric Colitis: the Seat of the Lesion and its Treatment.

By SIR JAMES CANTLIE, K.B.E., LL.D., M.B., F.R.C.S., Etc.

Surgeon to Seamen's Hospital; Lecturer on Surgery, London School of Tropical Medicine, etc.

COLITIS of dysenteric origin is a lesion frequently met with in Britain to-day. It has increased in amount since the war began, but before then it had relatively increased since emetine was introduced for the treatment of dysentery to the exclusion of ipecacuanha. The sudden influx of many soldiers invalided for colitis, during the past five years, was due to the large numbers of men campaigning where amœbic dysentery prevailed; but we have had in pre-war years a constant supply, although in smaller numbers, of men invalided by dysentery coming to our shores. When emetine began to supplant ipecacuanha, post-dysenteric colitis with its recurrences and persistency became noticeable, and the sudden multiplication of numbers has added to the evidence. Be that as it may, the presence of post-dysenteric colitis is an ailment we have frequently to treat in Britain at the present moment, and the treatment is so varied that it proves there is no finality to either our exact knowledge of the nature of the lesion nor to the proved efficacy of measures recommended.

The seat of the lesion is at the junction of the sigmoid flexure and the rectum, some eight inches up the bowel from the anus. The writer is fully aware that he is using an old-fashioned nomenclature when he speaks of the *sigmoid.* According to the modern anatomist there is no such entity as the sigmoid flexure; it has been divided into two parts, the upper belonging to the descending colon, the other (the lower or so-called pelvic portion) to the upper part of the rectum. The writer has in previous writings asserted that whatever advantage anatomists may claim from this change in nomenclature, it is from every other point of view, be it physiologically or clinically, inapplicable. He has elsewhere contended that the sigmoid flexure is an entity, and has to be considered as a whole, having distinct functions, structure, and pathological lesions. Shortly, these differential anatomical details are: (*a*) that this portion of the intestinal tract has a distinct beginning and ending; its upper end or inlet and its lower end or outlet are definite points, therefore he has named them the colo-sigmoid pylorus and the recto-sigmoid pylorus respectively; (*b*) the arrangement of the longitudinal muscular fibres of the intestine at this point resembles those which obtain at the gastro-duodenal pylorus—the pylorus, as it is named. The inlet is wider than the outlet into the rectum, and it is at the outlet that the post-dysenteric or chronic colitis lesions obtain. The mucous membrane of the lumen of the sigmoid differs in arrangement and in its general appearance from that of any other tract of intestine, and is quite characteristic and specific. Whatever the anatomist may say however, there is no gainsaying the fact that clinically the lower two inches of the sigmoid is the seat of the lesion met with in the ailment in question. This can only be determined by examination of the region by the sigmoidoscope. Without this, a concrete idea of the seat of the lesion is mere guesswork, the treatment of the condition empirical in the extreme, and the prospect of definite cure rendered uncertain.

The lower aperture or recto-sigmoid pylorus is endowed with as pecial sense. The passage of fæces through this aperture warns the individual that a stool is imminent; the fæces are stored in the rectum for a time, it may be, until convenience allows of a stool being passed; were it otherwise, a rush to stool would be necessary and momentary relief imperative. That this is the case, that the recto-sigmoid pylorus is the dial of defæcation as the fauces are the dial of thirst, is easily proved to even a careless observer, for: (1) When passing the sigmoidoscope up to and through the recto-sigmoid aperture the patient always exclaims, "Look out, I am going to have a stool"; (2) When, through a "colotomy" aperture above the left groin, a tube or bougie is passed downwards along the lower part of the sigmoid, towards the rectum, the moment the tube or bougie reaches the lower aperture of the sigmoid and is passing into the rectum, the patient makes a similar exclamation and declares he is going to have a stool. Here is the narrowest point of the great intestine, and in any inflammatory lesion of this portion of the bowel the lumen of the tube is relatively more acutely narrowed than any other. The effect of a colitis is more apparent here than at any other point. Any fulness here amounts to an obstruction, a strictured or narrowed point, and as the lesion persists it causes, as in a strictured urethra in the neighbourhood of the membranous portion of the urethra, post-strictural lesions of well-known clinical characters. In the urethra there is retention of a small quantity of urine, which undergoes decomposition, induces a chronic change on the bladder side of the tube which causes a gleet, and in time changes in the mucous surface of the bladder. So in the bowel, the obstruction in the lower end of the sigmoid induces a catarrhal condition of the upper part of the sigmoid mucosa, which, owing to the enormity of the tract of the bowel compared with the minute urethra, causes an overflow not of the minute dimensions of a gleet but of an avalanche of mucous secretions, the "mucus-in-the-stool," so definite a feature of bowel lesions.

Examinations of the bowel by the sigmoidoscope show yet another clinical feature of definite persistency. It is no other than that, in the dysenteric bowel, the lower end of the sigmoid flexure, the whole is prolapsed into the rectum to a greater or lesser extent. The sigmoid is normally always slightly prolapsed into the rectum, in much the same way as the uterus is naturally prolapsed into the vagina; but the thickened dysenteric bowel (the strictured condition, in fact) obtaining at the entrance to the sigmoid, causes the prolapse to become exaggerated and to increase until the prolapsed sigmoid can be felt in the rectum as a pear-shaped mass by the finger introduced per rectum.

It is the prolapse of the sigmoid into the rectum that is the cause of the tenesmus characteristic of dysentery, colitis and other cases of intestinal flux, for is not this the seat of the "defæcatory sense" as referred to above?

The treatment of these post-dysenteric lesions being within the view of the sigmoidoscope resolves itself in a surgical manipulation. This is the method adopted by the writer:—

(*a*) Introduction of the sigmoidoscope without an anæsthetic, thereby avoiding all possible sources of danger, for the patient's feelings indicate any danger point. Push the tube up as far as possible—that is, as far as pain will allow—withdraw the rod-stilette, dilate the rectum above the point by squeezing the air into the bowel from the air bag, and push the tube onwards exposing the mouth of the sigmoid. Wipe the surface gently with a piece of dry cotton wool held in the sponge holder.

(*b*) Inspect the surface exposed; there may be ulcers, excoriations, pinhead-sized hæmorrhages into the mucosa or a velvety and œdematous appearance of the bowel surface. The rectal mucosa adjacent may be normal or merely œdematous in appearance. If pain allows or the narrowed bowel permits, push the sigmoidoscope onwards until well into the sigmoid flexure, when, as a rule, a healthy surface will be found extending from two inches above the lesion at the lower opening of the sigmoid.

(*c*) Now withdraw the lamp, leaving the tube in. Dip the head of the rod-stilette into pure carbolic acid, push it along the empty tube, touching the affected surface at its highest point with the carbolic, withdraw one inch, and after withdrawing the rod-stilette and dipping it again into carbolic re-introduce it as far as it will go along the tube. The whole instrument may now be withdrawn.

(*d*) After two days' time, the bowel should be washed out by an enema of *salt water* every day for four days, then every second day for a week, twice a week for yet another week, and then once a week for, say, a month. The salt water should be *sea water* wherever that is available. The writer used, in pre-war days, to get it from the open sea, in either the Havre and Southampton route midway across or in the Harwich and Antwerp route. But a mile out at sea from our coast, except at the Thames mouth, will suffice. The water should be filtered through a towel and then just brought to the boil (really scalding the water (180° F.) is better). Instead of sea water a treble strength of normal saline will do, or the artificial sea water referred to in *Martindale, extra Pharmacopœia.* The addition of a tablespoonful of rectified spirit (or whisky, if it can be had) does away with the lowering effect the enema tends to produce.

(*e*) A teaspoonful or two of castor oil every night at bedtime is the only drug usually required. Should a relapse of mucus, or mucus and blood occur, the same treatment may be repeated. Should a relapse of true dysentery come on, treatment by emetine until the blood disappears should be given, followed, if necessary, by a 30-grain dose of ipecacuanha repeated for four consecutive days, taking at the same time the usual precautions, when ipecacuanha is given, to avoid vomiting.

(*f*) *Diet.*—Except when blood is present in the evacuations ordinary food may be taken, but only one course at a meal. Milk and milk puddings are to be rigidly excluded from the diet, as they should be in all bowel lesions, whether due to sprue or to any of the inflammatory affections. Should a fluid diet be indicated, rice water and not milk is to be given.

The Tests for Tuberculosis of the Lungs and for Consumption.*

By H. BATTY SHAW, M.D., F.R.C.P.
Physician to University College Hospital, and to the Brompton Hospital for Diseases of the Chest.

THERE can be no necessity to apologize for discussing this subject, because it is clear to us all that it is of such widespread interest and that it concerns each one of us—medical men personally—as well as our patients. It is not only a question dealing with suffering humanity, but also with those who are apparently quite normal in health. The whole fabric of any scheme for successfully coping with tuberculosis must be based upon pathological knowledge, if anything really useful is to be attained to.

Tuberculosis of the Lungs versus *Consumption.*—These two pathological states are not identical. Tuberculosis of the lungs, including the lungs proper, the pleura, and lymphatic glands, is a disease over which we are still much puzzled. We know that in civilized communities each individual is exposed to the risk of infection very early in life, and that each individual with varying success resists the spread of infection from the initial deposit, *e.g.*, the focus of disease may become encapsuled and rendered relatively harmless, or the actual organism may die and be destroyed right out. Unfortunately, we also know that the infection may pursue a rapid development and spread until, at some time subsequently, the individual reveals a lesion which, may be, expresses itself by a symptom first, or by a physical sign first, and this lesion may be so serious as to destroy a patient in a few weeks after such development. But fortunately we know that scores of individuals just as certainly may only reveal a slowly progressive lesion—a lesion which after a stormy onset may become less stormy and even lead us into the belief of its being cured, only to startle us at a remote date by the reminder that it was only dormant. Lastly, it is common knowledge that though a cure of this disease is not effected, *i.e.*, though the signs and symptoms may persist, through a long life, the individual's usefulness is but little impaired.

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* A lecture delivered at a meeting of the Bromley Medical Society.

All these various aspects of the disease called tuberculosis of the lungs have one of the most definite pathological backgrounds. The lymphatic glands of the naso-pharynx, trachea, or bronchi, may entrap the bacillus, the bodily economy may not suffer, because the glandular defence is effective—the organisms are destroyed by what we must remain satisfied to call "resistance"; we may not be able to see the effect of the struggle, except by the appearance of a special sort of giant-cell in the lymphatic gland, or by a caseating focus in the interior of the gland, or by the occurrence of calcification of the gland. All these satisfactory evidences of victory are accompanied by varying degrees of fibrosis. The tuberculosis may reach the apices of the lobes of the lungs, having encountered the checks of the above-named lymphatic glands, but may at the new site undergo the same cure. The same happy change may occur after tuberculosis of the pleuræ, even when accompanied by effusion.

The infected individual does not always master the infection. Well we know the sorry results—

tuberculous adenitis of the neck with life-long scars, following spontaneous or surgical relief; tuberculosis of the mediastinal glands with involvement of the lungs or pleuræ by extension; tuberculosis of the lungs beginning at the apices and spreading by extension to the rest of the organs, or lymphatic or vascular infection producing universal tuberculosis, or the tuberculosis may spread by aspiration from one infected area to a hitherto healthy one. Too often death results, but we know that even death is postponed in some of these cases until a late date, and may be brought about by some other infection altogether. In such cases, fibrosis with arrest was not an early solution; it may come later and be effective, it may be present all the time but be ineffective.

What are the tests of the above pathological conditions? I want you to keep in mind that we are for the moment dealing with a pure tuberculous infection, with a process in which the miliary tubercle is the first and earliest reaction to the tubercle bacillus; this lesion to the end is the only sign of activity. In other words, the lesions underlying all the above are essentially small; they may be few—they may be many—in some cases a few may join together and form larger foci of disease—the tubercle has become conglomerate and the lung may reveal small patches of consolidation. In rare cases the tuberculosis may not be recognizable as such, and the infection may assume the features of massive confluent pneumonia. In many cases the tuberculosis gives place to small fibrotic scars, to scar-like calcareous masses so familiar at the apices of the lungs of otherwise non-tuberculous subjects. In other cases the fibrosis is enormous, and the lungs are scarred and contracted almost out of recognition. What I want to insist upon is, that suppuration and cavity formation form no part of the tuberculous infection. The tubercle bacillus does not cause suppuration, at any rate directly.

Now, what is consumption? To most of us it is the stage of tuberculosis of the lung in which there is *consumption or loss* of substance of the lung, with cavity formation, with copious purulent expectoration, and all the symptoms of septicæmia, especially wasting, *i.e., consumption of the individual.* This view must be a cardinal feature of our discussion to-night. Personally, I do not think consumption is tuberculosis of the lungs. I think it is due to infection of the damaged lungs by pyogenic organisms. Pathological studies point to this theory most conclusively. The expectoration of a consumptive patient may teem with pyogenic organisms as well as showing many (or a few) tubercle bacilli. Purulent expectoration is caused by lesions of the lungs due to the pneumo-coccus, in the absence of the tubercle bacillus. An infectious "cold in the head" is frequently followed by bronchitis and broncho-pneumonia and purulent expectoration. Excavation of the lungs occurs in the repeated absence of the tubercle bacillus from the sputum, and the post-mortem shows excavation of the lungs independent of tuberculosis. I would also remind you how rare it is for a primary tuberculous pleural effusion to be purulent; it is a serous effusion, and remains so till absorbed. Hæmoptysis is a feature of non-tuberculous excavation of the lung. I have seen just recently a ruptured aneurysm in a large cavity of the lung; the hæmorrhage caused the death of the patient, but the sputum during life was free from tubercle bacilli, and the most diligent microscopic search through the walls of the abscess cavity and adjacent lung showed no trace of tubercle bacilli, or even of giant cells.

Although it has been found that so many children who succumb reveal tuberculous lesions in some organs or tissues, the same has not been shown with regard to suppuration in the same tissues, and numerous as may be the cases of tuberculosis in dead young children, consumption in them is comparatively rare; that is a complication which comes on later, and I think we should all agree that the so-called respiratory catarrhal infections are the agencies by which pyogenic organisms are introduced into the lungs. Reduce our catarrhal colds, and we may look for a reduction of consumption.

Active Tuberculosis versus *Inactive Tuberculosis of the Lungs.*—We must now endeavour to distinguish between cases of active and inactive pure tuberculosis of the lungs. I think it will solve a part of this difficult problem if we admit at once that all town-dwellers at least are examples of "reacting" carriers of the tubercle bacilli. We must qualify the term carrier, because the term unqualified has a different significance, and means that an individual is carrying an organism in some organ or tissue, that it is not doing the individual any harm and is not even provoking a reaction on the part of the neighbouring tissues of the individual. We are all familiar with individuals who are carriers of the pneumococcus, the typhoid and diphtheria bacilli; comparative pathology shows, too, how one species of animal may (for a time at least) be a carrier of an organism which, gaining access to members of another species, may produce death very quickly.

A "reacting" carrier, in the sense of the expression which I have used, may react so completely as to destroy the organism which has caused the tissues of the carrier to react; we should expect to find, in such cases dying from some other cause, that the tissue reactions are the usual and characteristic ones, and that all biological and microscopic tests fail to show the presence of living tubercle bacilli. We also know that such lesions may show that the bacillus is not dead, and is capable of causing the death of a guinea-pig, on inoculation, from diffuse tuberculosis. Is there any means of telling when this "reacting" carrier is beginning to suffer from tuberculous disease? Has anyone yet defined the moment when we pass from being healthy "reacting" carriers to unhealthy tuberculous patients? Does the tuberculin test help us? I think you can answer that question in the negative at once, because no one of us, so far as I know, has determined how long the anaphylactic state, upon which the tuberculin test depends, takes to develop after the access of tubercle bacilli to a non-tuberculous animal or human being; and still more important is it to admit that no one has shown how long such hypersensitiveness lasts. With such imperfect knowledge upon these two points, it is clear that a positive tuberculin test may express a tuberculosis which is *over and done with* or it may express a tuberculosis which is *active.* It is of no use to look for tubercle bacilli in expectoration, urine, or stools in such cases, because the tubercle bacilli may be destroyed or shut up in tissues or organs which render such liberation of bacilli impossible.

To be brief, we are thrown back upon symptoms, *i.e.*, upon signs of functional disturbance, and if such symptoms are not found to be explainable by the presence of some quite different disease, we should suspect tuberculosis. What a very simple business it seems to be! I will now hand round to you a list which shows what weight you should be for your height and age. You know well that if your weight is below a certain margin which differs somewhat with different insurance offices, you would not, all else being normal, be accepted at ordinary rates. Hitherto such underweight has been largely of moment only from the insurance point of view. But we must admit that it is very important from the diagnostic point of view as well. It may be that some of you will tell me, later on, that you personally have always been underweight, that other members of your family have always been spare people, but contrive to work throughout life, even to a ripe old age, and have never been ill. You will be prepared at once to debate on such a point as this and say that, just as in the case of dietaries, we do not, from the physician's point of view, want to know what is the maximum or minimum of food of various kinds that is necessary for the individual, but what is the optimum, and you will say in regard to this question of weight that it is a more reliable method to depend upon the comparison of the present weight of a patient, with the highest weight of the same individual.

I am sorry to say that is not the "insurance," way of looking at such matters, and, whether you know it or not, it is not the way other members of our profession are viewing the matter. Underweightness is becoming a talisman for the discovery of what, in the absence of any other cause, must be the passage from the condition of a mere "reacting" carrier to the state of a sufferer from tuberculosis—somewhere in the body. I will say at once I agree with this view, but with a very big proviso—there must be nothing else which could lead to the wasting. When we think what such a close study of the individual this means, we can see that not only must a medical man be armed with a high, very high, order of technical skill in examination, but he must be a man, too, of very broad interests in diagnosis, and indeed he must be a man of the world too. I will spare you a rehearsal of all the pathological states which can cause wasting and which may reveal themselves, or may not, in a careful history and examination. I will also spare you a discourse on the subject of how disturbed mental states can beget a condition of leanness.

We must come, however, to the case of the man, woman, or child who is underweight and has no other symptom nor signs of any disorder, except perhaps that he or she has a cough. I can assure you that a diagnosis is frequently made of tuberculosis of the lungs from these two features, with almost assuredly some such alleged physical signs as slight dulness or bronchial breathing at that "area of romance," the upper part of the chest, especially the right one.

In the case of rich and unoccupied patients, it is not a material question whether such an individual is sent to a sanatorium in this country, or preferably to some warmer and drier climate than ours. But I must plead here against the practice of putting cases of this kind, who are earning a living or who are not rich and who are suspect only, to the expense of a treatment which is costly and far beyond the means possessed, and to the hardship of unemployment and removal to a distance from home and friends. Is there any reason why you should not keep such cases under observation, doing your best to increase the weight and remove the cough? You will not, by doing so, deprive anyone of the cure for pulmonary tuberculosis, even if the disease is present, for such cure does not exist, outside the patient himself. It looks as if starvation is at the present moment, amongst the children of Central Europe, leading to the conversion of "reacting" carriers into cases of pulmonary and other forms of tuberculosis. Feeding up is no prerogative of the sanatorium, it can be done at home.

It only remains here to be said that anorexia, night sweats, fever which is unexplainable, anæmia, and amenorrhœa apparently not due to some other cause, will be accepted by you as proof that the above patient is still more probably tuberculous. I hope, however, that you do not notify such cases, for it is well known that such cases under treatment will pick up, and apparently recover from their bad symptoms, even if they are, as I fully admit is possible, due to a temporary activity of the tubercle bacillus.

Now it is a reasonable question to put forward, viz., what *are* the signs of active tuberculosis of the lungs? It is quite clear from what has already been said that we cannot rely upon the presence of tubercle bacilli in the sputum—for with miliary tuberculosis there may be no sputum. We have seen that the tuberculin test is valueless, and quite rightly this test is not allowed to count by the various public authorities, who determine whether a case is or is not really tuberculous, when such case has been referred to them. Even provocative doses of tuberculin cannot be safely relied upon. We are again thrown back upon pure clinical investigation—prolonged, irregular fever and other symptoms already mentioned, which may be associated with changes in the normal physical signs of the lungs.

It is no time to talk to you of what those signs are. But it is a time when I should warn you against those signs which have been adduced as indubitable proof of the presence of tuberculous disease in the lungs. You will remember we are discussing the common condition, viz., miliary tuberculosis of the lungs—not consumption. I think you can only rely upon the discovery of the group of symptoms, etc., fever, breathlessness, and, in addition, a cyanosis which is not dependent upon any other cause, and possibly of cough; miliary tuberculosis of the lungs of a generalized character can only be detected in this way. If there are but a few miliary deposits at the apex of a lobe, or lobes, you cannot diagnose it at all.

It will not be a common experience to find cases of the massive confluent pneumonia which I have already mentioned, and of which I show you a specimen. The case is, however, easier when irregular temperature is accompanied by signs of retraction of the lung and deformity, or defective action of the chest wall—which we meet with in fibroid phthisis, so called. Every one of you has such a case under your care; they are familiar in out-patient practice in the spare, underweight man or woman—vigorous enough to work, though not able to work so hard, or so long as others—even incapable of manual work at all, but useful helps, nevertheless, to others who can do so; tubercle bacilli are rarely present in the expectoration, or they occur in sparse numbers at long intervals. The cases I refer to are rare under 40 years of age. They do not usually show many physical signs; indeed, often enough only those of emphysema, and perhaps defective movement at one or other apex, to those gifted with remarkable percussional powers, slight dulness is observable; the breath sounds at one or other or both apices are bronchial in type; apical râles cannot be detected. I think I am right in saying that you will not find such cases in the sanatoria devoted to the treatment of the poor; for I think only too often, at the age at which such a state is commonly found, they are not considered "suitable cases"; they are relegated to our out-patient departments, or to a dispensary, to live under "home conditions." Personally, I consider that by so dealing with them this class of case is well treated; they miss nothing by such relegation to the dust-heap of home life, until the time comes, alas, when work is impossible, and then I think the treatment meted out to them is disgraceful in the extreme—10*s.* a week from the State coffers, without adequate nursing!

So far we have discussed—

(1) "Reacting" carriers of the tubercle bacilli in the body, possibly including the lungs and adnexa.

(2) Active tuberculosis of the lungs.

(3) Inactive tuberculosis of the lungs, *i.e.*, cases that have shown signs which suggest that inactivity has followed upon activity of the tuberculosis, and some of the group become cases of—

(4) Fibrotic lungs or fibroid phthisis.

In these cases, the crucial test of tuberculosis of the lungs, viz., the presence of tubercle bacilli in the sputum, cannot be obtained, or at long intervals only, and then in very few numbers. Though no part of my programme, I should like here to express a wish that subsequently you will debate what should be the proper form of treatment of these cases. At the present moment many such cases receive the whole gamut of experiences granted by that part of the Insurance Act which deals with tuberculosis; yet we hear on all hands that there is no decrease of tuberculosis, that the mortality is increasing despite the Act, and we know that five millions have been spent on the attempts to cure tuberculosis since 1912.

We have no tests for active tuberculosis, other than functional disturbance; and when I admit this, I must repeat once more that every care must be taken, before a diagnosis of tuberculosis is made, to exclude other diseases and conditions which may lead to the same functional disturbance.

Tests for Consumption.—We have at long last reached the cases which by some are also called tuberculosis of the lungs, but which should assuredly be called by some name which expresses all the truth, viz., that the tuberculosis of the lungs is accompanied by destruction of the lung tissue, brought about by "mixed infection." Even if tuberculin is a cure for tuberculosis of the lungs, it is bound to fail, as it has done, to cure "consumption." Even if the open air methods known as sanatorium treatment cure pulmonary tuberculosis, they are bound to fail in this country, as they have done, to cure consumption. Indeed, I am sure that some cases of consumption (and of tuberculosis of the lungs) are the worse for open-air treatment, so called. The reason is not far to seek, because the exposure to open-air conditions of life in this cold, damp climate causes pulmonary and bronchial catarrh.

We have a rule at the Brompton Hospital which says that only those cases which reveal tubercle bacilli in the sputum are to be notified. I think this is a good rule, because it is only such cases amongst those who are suffering from consumption which are a source of danger to the young children in their neighbourhood. For a long time I used to label only those cases as pulmonary tuberculosis which revealed tubercle bacilli in the sputum. I now label them as cases of consumption, because I think by so doing I shall bring my treatment more into line with the true pathology of tuberculosis of the lungs on the one hand, and consumption on the other.

Is it a straightforward problem to diagnose consumption, or has the disease its own difficulties such as we have seen pulmonary tuberculosis has? Of one thing I am sure, that such classical signs of consumption which used to be advanced, viz., those of cavity formation at an apex, or prolonged fever with consolidation of an apex, are not invariably due to the tubercle bacillus accompanied or unaccompanied by "mixed infection." As I have already hinted, other organisms which affect the respiratory tract are able to produce the same signs, and even such a striking symptom as copious hæmoptysis. Such cases are not frequent, but sufficiently frequent to put us on our guard to insist that, before we notify a patient as dangerous to the community because of the risk of infection by the tubercle bacillus from the sputum, before we subject our patients to the extremely unpleasant form of cure which is said to cure tuberculosis and consumption, we should insist upon several examinations of the sputum, one of which should be the examination of the residue of sputum after chemical digestion, for tubercle bacilli. If tubercle bacilli are not found, do not notify.

PROPHYLAXIS.

Having said what I have said, to try and point out the tests for what I think are two different conditions, viz., pulmonary tuberculosis and consumption, I hope you will not consider that I came amongst you merely to discharge a more or less academic task. You may say that after all, whether a case is one of pure tuberculosis of the lungs, or whether it is one of tuberculosis and mixed infection, *i.e.*, consumption, there is no new lesson for the practical management of such cases. That would be a mistake, and if I did not think so I should not have come to address you to-night.

As a result of the Insurance Act and its numerous agents, such as health visitors and tuberculosis officers, and as a result of notification, we may reduce the amount of infection of young children with tuberculosis, and, as a result, civilized humanity may not in the future be so widely affected with tuberculosis of the lungs. But if we are to check the much more serious condition, consumption, we must prevent respiratory catarrhs more. Greater attention must be paid to abnormalities of the nose and pharynx; further, we must accept the doctrine that prophylactic vaccination against catarrhal affections of the respiratory tract has come to stay.

In your company some years ago I did my best to refute the doctrine that vaccination was of any specifically curative use, when disease had established itself; I still hold that opinion. I was not hostile then —I was even favourably inclined towards prophylactic vaccination—and I still am a believer in such treatment. If you have a patient who comes of tuberculous stock, who reveals signs or symptoms which show they are liable to respiratory catarrh, I am quite sure you should use prophylactic vaccination, preparatory to the onset of our season of colds and catarrhs. If your patient is not relieved of attacks of catarrh by such methods, I am quite convinced that you should do your best to persuade him to leave the country and seek a livelihood in those parts of our Empire which have warm, dry climates. No other country, state, or dominion would shut its doors against such patients; if you wait till consumption is manifest, in however early a stage, you will find that such emigration is impossible; local legislation puts restriction on such action.

Our rich and well-to-do are leaving these islands now for the south of France and elsewhere, where the climate is more salubrious. Our poor, who have tuberculous tendencies, are condemned to stay here, too often living in slums and working in unhealthy business places. I am hopeful that our new Ministry of Health will carry on such propaganda as I have put before you to-night, in favour of emigration for these candidates for consumption before it is too late; and I hope the Ministry will get more quickly to work to provide decent houses for the poor, so that the "reacting" carrier, who, like the poor, will I fear, always be with us, shall not become (1) a case of active tuberculosis of the lungs or other organs; or (2) a case of consumption.

Medico-Legal Notes

(*continued*).

By SIR JOHN COLLIE, C.M.G., M.D., J.P.
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MEDICAL REPORTS AND THE LAW OF LIBEL.

MOST medical practitioners are uncertain, and therefore nervous, about the extent of the responsibility they incur when furnishing a report upon the result of an examination made for medico-legal purposes. It is desirable, therefore, to have a clear understanding as to how far it is safe to express an opinion about an injured person's physical condition, his mental attitude towards his work, and how far he really desires to recover fully. Whilst we are all anxious to do our duty, no one desires to risk an action for libel or slander—even if he is a member of one of the two great Medical Defence Unions.

What is "libel," "slander," "defamation"? W. Blake Odgers, K.C., Recorder of Bristol, in his well-known work on the *Law of Libel and Slander,* states: "Words which produce any perceptible injury to the reputation of another are called defamatory. False defamatory words, if written and published, constitute a "libel"; if spoken, a "slander"; and, undoubtedly, statements that a man is feigning or exaggerating would fall within this category. But the learned author points out that *in certain circumstances* one has the right to state plainly and honestly what he believes to be true of another, and to speak his mind fully and freely concerning his character. This, however, must be done under what are known as privileged circumstances.

There are two kinds of privilege: absolute and modified. What is said by a judge is protected by what is known in the law as absolute privilege. What is said by a witness in the witness-box is covered by the same absolute privilege. This class of privilege is also extended to what transpires in the confidential relationship which exists between a solicitor and his client; and in confidential relationships where a duty is thrown upon one person of protecting the interests of the other, it is clearly not only excusable but imperative that he should be privileged in expressing his *bonâ fide* and honest opinion.

Clearly a person against whom an action is brought, or even threatened, for personal injuries alleged to have been sustained—and the more so if, in fact, he has to defend one—has a right, if so advised, to set up the defence that the plaintiff is not so seriously injured as his statement of claim alleges, or even that no disability exists; and for the purpose of obtaining advice on such questions he is entitled to know from an experienced medical man if, in his opinion, the person who so claims has in fact been injured, and, if so, to what extent. Any opinion expressed by a medical man to the party concerned, his legal representatives, or authorized agent, for the purpose of advising him about his position with regard to a defence, is the subject of modified privilege, which becomes absolute provided the opinion is given *bonâ fide* and without malice. Therefore the report of the medical man, and, of course, his statement of the evidence he is prepared to give, under these conditions, becomes absolutely privileged; the circumstances, and the occasion, afford an absolute bar to any action at law in respect of them.

It cannot be stated too plainly that when a medical man reports, either verbally or in writing, upon an injured person, where there is or may be a dispute, he is fully at liberty to state quite freely, and in as emphatic language as he chooses, what he *honestly* believes. The one all-important point to remember is that the verbal or written report must on all occasions be furnished exclusively to those who are entitled to it. The only danger is in giving an opinion to those who have no right to call for it. Summarized it comes to this: medical certificates or reports furnished *bonâ fide* and without malice to a party to an impending dispute, or his legal advisers, are of a confidential nature, and therefore privileged.

Only in very extraordinary circumstances could it be suggested that a medical man had imported malice into his report. It is conceivable that, in a case of which he had previous knowledge, and antipathy to the person he was examining, he might use language which not merely evidenced his dislike, but showed he was actuated by malice, in which case he could not claim privilege, and rightly so. If, to call a man a malingerer, to say that he is obviously and intentionally untruthful, that he is in perfect health whilst claiming large damages for total incapacity, constitutes libel, then almost daily for many years I have laid myself open to actions for libel. The law, however, is so well settled that, by a due observance of the conditions imposed, I know I am immune.

Medical officers of institutions frequently supply information about inmates who are, or have been, under their care, without first obtaining the permission of the patient to furnish the report asked for. Not only is this ethically wrong, but in such a case privilege could not be claimed. The fiduciary relationship in this case is between the doctor and the patient, and cannot be transferred except with the consent of the patient; and it is on fiduciary relations that the doctrine of privilege is based. It should be remembered that an inmate of a hospital, infirmary, or similar public institution, submits himself freely to examination, gives a full account of the happening of his accident, etc., solely for the purpose of assisting the medical officer to effect a cure. Information so obtained must not be communicated in a report to a third party, without first obtaining the consent from the injured person concerned. An action brought in respect of such a report could not be defended on the ground of privilege; but, on the other hand, provided the patient fully understands the object with which the report is made, and gives the medical officer of the institution permission to furnish the report, it is perfectly correct and safe for the medical officer to state frankly and impartially his opinion of the case, even though such report may be to the detriment of his patient.

RECENT ALTERATIONS IN THE LAW.

The following alterations of the existing Workmen's Compensation Acts and of the Common Law may be of interest to those who from time to time appear in the Law Courts.

In August, 1917, the Workmen's Compensation (War Amendment) Act, 1917, was passed; it came into operation on September 1, 1917. It provided that compensation payable to an injured workman under the Act of 1906 in cases of *total* incapacity (whether the incapacity arose before or after the commencement of the Act) should be increased by *one fourth.*

It will be observed that the increase in the amount of compensation, *i.e.* the weekly payment, only relates to workmen *totally incapacitated* and to such as come under the Act of 1906.

An Act to amend further the above-mentioned

Act came into operation on January 1, 1920. It provides that instead of the increase in the weekly compensation being one quarter of the original amount, *i.e.* 5*s.*, it is now three quarters, *i.e.* 15*s.*; making a maximum of 35*s.* per week.

In July, 1918, an Act (the Juries Act, 1918) was passed, which came into force immediately upon passing. This provided that every action in the High Court should be tried by a judge alone without a jury, with, however, the following exceptions :—

(*a*) Cases in which libel, slander, malicious prosecution, false imprisonment, seduction or breach of promise of marriage are alleged, and either party to the action is entitled as of right to a trial with a jury.

(*b*) If either party to an action satisfies the Court that the question involved is more fit to be tried with a jury, the Court may order it to be so tried.

(*c*) Certain cases under the Matrimonial Causes Act, 1857.

In County Court actions, neither party can require a jury to be summoned to try the action if the amount claimed does not exceed £5, unless the case involves matters set out in (*a*), (*b*), or (*c*) above; but the judge may, if he thinks fit on the application of either party to the action, order it to be tried with a jury.

The age of jurymen liable to serve was extended from 60 to 65 years.

All these Acts are to have effect until six months after the termination of the war. By the Termination of the War Act, 1918, the date of the termination of the war is to be declared by an Order in Council, and the date to be fixed for that purpose is to be as nearly as possible the date of the exchange or deposit of ratification of the treaties of peace.

(*To be continued.*)

Dislocation of the Shoulder-Joint and its Treatment.

By ALAN H. TODD, M.S., B.Sc., F.R.C.S.

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THERE is no civil accident that is at all common at the present time, which is more unsatisfactory, as regards the ultimate functional results, than dislocation of the shoulder-joint. Diagnosis and reduction are usually good enough, but the subsequent treatment is altogether bad. This unfortunate fact is partly due, no doubt, to the common practice of transferring patients to a massage department, where the patient comes under the care of someone else, so that the original surgeon never follows up his cases. But the main obstacle to progress is the slavish adherence to traditional methods, that we have bewailed above.

If we take the trouble to follow these cases up, we shall be shocked to find that many of them suffer prolonged disability; in some, partial disability is permanent; in nearly all, the course of treatment is unduly long. This means much pain endured, much time lost, and much money wasted. The records of any big insurance company will go far in support of this statement; at one such concern alone, the writer was informed that cases of dislocated shoulder cost about 4,000*l.* a year, and that the average time which the patient spent on the pay-roll was *twelve weeks!* This includes a large number of patients, it must be remembered, coming from a very wide area, so that they represent the common result of treatment, and not the practice of any one hospital. The result can only be described as deplorable, and is a very grave reflection on our present methods of treating the condition.

The writer had an exceptional opportunity of studying these cases in the earlier part of the war, when acting as surgeon to the Fracture Out-patient Department of Guy's Hospital, during the absence on military service of Mr. L. Bromley. The first thing that struck him was the very great difficulty that was experienced in restoring the power of abducting the arm, after the patients had been treated in the ordinary way, *i.e.*, by bandaging the limb to the side for a week or more after reduction. In the first instance, no doubt, the limitation of movement is due simply to pain, for if the limb is moved with extreme care, abduction to a right angle can be obtained without difficulty; but after the lapse of a few days, a mechanical obstacle appears, in the form of adhesions about the lower part of the joint. When first these begin to form, they can be stretched, or ruptured, by fairly forcible passive movements. But it must be remembered that most of these patients are elderly, and will not submit to such treatment; moreover, violent manipulations in patients of this age usually set up so much pain and swelling, that they either absent themselves from hospital altogether or else one is compelled, out of pure humanity, to allow them a few days' rest, and during this time the joint becomes as stiff as it was before, or even stiffer. Again, these people are either actually osteo-arthritic or else they are predisposed to the condition by their age and constitution, or by their oral sepsis; violent movements are to be deprecated, as being likely to set up osteo-arthritic changes, even if the accident has not done so already.

The outcome of this was that a trial was made of putting up one or two cases on a Middeldorf triangle, instead of bandaging them to the side as usual. This seemed to be an improvement; abduction was more easily restored, and no disadvantage was apparent, beyond the awkwardness of the lower end of the splint when lying in bed. The idea was therefore developed, and the arm even more freely abducted from the side; the easiest plan was found to be that of resting it upon an ordinary rectangular wooden splint, one limb of which was strapped to the trunk, whilst the other supported the arm. The tendency of the splint was of course to sag down, so that the abduction became less than a right angle, but this can be combated to a certain extent by putting a long piece of strapping right round the splint and over the opposite shoulder, in addition to the pieces which fix the splint to the thorax and to the arm.

A more comfortable apparatus by far, and one which has no tendency to slide round the chest, consists in a half-jacket of poroplastic felt, or non-inflammable celluloid, or (best of all) of *papier mâché*, to which is fixed at right angles a trough to support the arm and another trough to support the forearm, with a hinge-joint at the elbow. Such an apparatus was made by the Surgical Requisites Association during the war, under the name of the Acheson Splint, and a most admirable splint it was; we were fortunate in having two of them at Guy's, and they were used for several of these cases with great success. Another simple splint, easily made by any blacksmith or tinman, consists of a large piece of sheet-metal curved to fit the chest, and a smaller one to fit the arm, joined together by a rod of stiff hoop-iron.

The exact pattern of apparatus used, however, is immaterial, provided that rectangular abduction is achieved from the very first. In no single case did we find it impossible to obtain the desired abduction, provided that the splint was applied within two days of the reduction of the dislocation; and if the case had been seen by us at the very outset, rectangular abduction would have been employed from the moment when the dislocation was reduced, under gas. If the patient has had his arm bound to his side for some days, it will be necessary either to abduct it by degrees or else to give gas, and abduct it straight away; on the whole, the latter method is preferable, since it is neither painful nor risky, and it greatly shortens the period of disablement.

It will be remembered that there are no muscles protecting the inferior aspect of the shoulder-joint; that the capsule there is thin, and thrown into redundant folds, especially when the arm is adducted to the side, and lastly, that the tear, in dislocation, is always at the inferior part of the joint. If the arm is tied down to the side, in the after-treatment, it is more than likely that the torn edges of the capsule will be crinkled up and adhere to the humerus, or to the folds of the capsule, whilst these folds will be covered with an inflammatory exudate, resulting from the traumatic inflammation, and will stick to one another in a knobby mass. It is this mass of adherent, crinkly capsule that constitutes the obstacle to abduction, and in old cases it is so tough that it absolutely binds the arm to the side, and will not allow of more than 90° of abduction at the very outside.

If any further abduction of the arm is desired, it can only be achieved by a rotatory movement of the scapula over the chest-wall, the angle between the long axis of the scapula and that of the humerus remaining unchanged. It is very important to grasp this principle; the scapula can rotate over the chest-wall through a right-angle, but no more; if, therefore, the arm is fixed at a right-angled abduction, and grows stiff there, it can be raised above the head by rotating the scapula through 90° in an outward direction, while it can be brought to the side by rotating the scapula through 90° in the opposite direction. If, on the other hand, the arm has been fixed down to the side, the utmost possible movement will be through 90° of abduction instead of 180°. Moreover, it is obvious that time is gained by starting treatment with the arm already at 90° abduction, instead of nil; we have then only a further 90° to restore, instead of the full 180°, in order that our patient may be able to hold his arm above his head. Further, much pain is avoided, for experience shows that it is fairly easy to get the first 90° of abduction back, but that the next 90° are only obtained very slowly and with much suffering. Indeed, in many patients, they are never obtained at all.

Abduction offers the best possible chance of bringing the torn edges of the capsule into accurate apposition. When the arm is brought down to the side, the capsule is crinkled up into folds; when the arm is abducted, the capsule is put on the stretch; it follows, therefore, that the rent in it is stretched out also, and therefore its edges must be brought into apposition, just as the two sides of a rubber ring are drawn together, if it is pulled upon from opposite ends. Again, in abduction, the rent is closed by the tension of the capsule, whereas in adduction it is slack, and open.

In abduction, the muscles which extend from the trunk to the arm brace the humerus firmly up against the glenoid cavity, whereas in adduction they are more or less relaxed, and the head is less firmly held.

The majority of the text-books in common use to-day advise that the arm should be bandaged to the side after reduction; one of the latest and best says that after 48 hours the joint should be "gently moved, care being taken not to abduct the arm. . . . Active movements should be begun at the end of a week. . . . The movement of abduction is the one that should be practised last. . . . Six weeks should elapse before the joint is used freely." All the books agree that the arm should be fixed to the side at first; some say for a few days only, with massage in the intervals, whilst one still advises a fortnight's immobilization. This, we contend, is radically wrong; it is wrong in theory and anatomically bad, as we have explained above; the results prove it to be bad, and (most important of all) *practical experience* has shown that the results of the abduction method are far better, that they are more rapidly obtained, that the method involves less pain, and that it is free from risk.

The only possible reason that can be advanced in favour of fixing the arm to the side is the desire to avoid a re-dislocation during the healing of the rent in the capsule. It may be stated at once that not one case of re-dislocation has been observed amongst the patients treated by the abduction method, whereas amongst those who had previously been treated by the old method, there were two who had had chronic recurring dislocation ever since, though their shoulders were quite normal until the time of the initial accident. As a matter of fact, the risk of re-dislocation is less, when the arm is abducted to a right angle than when it is by the side, for in the latter position the rent in the capsule is gaping open and slack, whereas in the former position it is tense, and its lips are in close apposition and likely to heal well. In abduction the muscles brace the humerus against the glenoid cavity, whereas in adduction they do not, or rather, they do not do it so effectively. It is only in hyper-abduction that there would be any risk of re-dislocating the shoulder, and this we do not advise at first. It must always be remembered, moreover, that the force required to dislocate a shoulder is excessive; no ordinary violence will do it; it is in the highest degree improbable that an arm that is supported on a splint, in rectangular abduction, would ever be exposed to a blow from above that would be strong enough to force the head of the bone down into the axilla.

The main point, however, that we wish to urge is, that in actual practice the abduction method is found to give better results than the old method, with less pain, no risk, and less expenditure of time, which is money. Theory and argument have their uses, but actual results are the best test of all. Anyone who will make an impartial study of the results of the older methods, and compare them with the results that he can obtain by following the directions here laid down, can satisfy himself of what we say.

In this connection it is interesting to note the remarks of Scudder, of Massachusetts General Hospital; he writes :—" It is customary to immobilize the reduced shoulder for many weeks without giving it any passive motion. It is my experience that poor results follow such treatment. It is far wiser and safer to make gentle passive motion upon the first day after the reduction and to continue these gentle movements with increasing force and exertion each succeeding day, until at the end of a week or a week and a half the patient is no longer restrained in his movements, but is encouraged to make all movements that are natural." This was written in 1911, and shows a great advance upon the treatment that is commonly used in this country, even to-day; but we go even further than Scudder, and advocate abduction from the very first, though we differ in advising *rest* in abduction for the first week or ten days.

Our standard practice is to abduct the injured limb immediately after reduction, or at any rate, as soon as we see the patient in the Fracture Out-patient Department, and to support it in rectangular abduction upon some sort of splint; the patient is told to attend daily at the Massage Department for massage, for there is usually considerable bruising and effusion; the treatment greatly decreases the pain and sleeplessness. No movement is allowed at the shoulder at first, but the rest of the limb is moved about freely. After 10 to 14 days, the splint is removed; the arm falls to the side without any difficulty whatever; the patient is told to carry it in a sling for about a week more, to perform fore-and-aft swinging movements, to rotate the arm externally and internally, and to practise abduction daily; the range of abduction is steadily increased, but always by steady effort, and never by any violent wrenching movement. A good plan is to make the patient try to reach a little higher, every day, up a wall, marking the level reached on each occasion. It will be found that all movements below the level of the shoulder are readily performed, whilst difficulty is experienced with the over-arm movements. Circumduction is the last thing attempted. The patient is not discharged until he can lift his hands quickly and painlessly up into the air, and interlock his thumbs above his head.

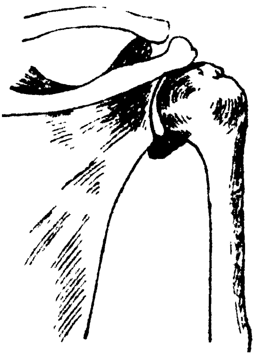

FIG. 1.—*Redundant lower part of torn capsule in abduction. Folds adhere to one another, and to humerus, and thereby limit abduction, and cause pain.*

In order to ascertain the end-results of some of these cases, a letter was written to all the patients who had been treated for dislocation of the shoulder at the Fracture Department between November, 1916 and the end of May, 1919. There were 52 in all, and 19 of these came to the hospital and were personally examined by the writer. The average age of the men was 53¼ years, and that of the women was 43½ years. With one exception, all had been reduced on the same day as the dislocation, and all had had subcoracoid dislocations. Thirteen men and 6 women were seen, and of these, 11 had left-sided dislocations, and 8 had right-sided ones. Only in one case was there any evidence of nerve-injury, and that was the case of an old and infirm man of 77, who had his circumflex nerve torn at the time of the initial injury; he was treated in abduction, but in spite of much electrical and other treatment, he still has complete reaction of degeneration, and shoulder-drop, with absolute loss of the power of active abduction; it is interesting to note, however, that abduction can be carried out, passively, to the full extent, which shows that there are no capsular adhesions to prevent it.

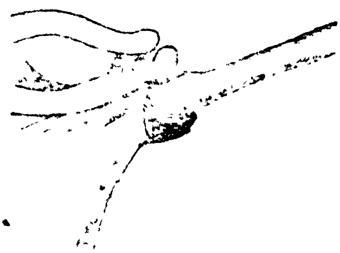

FIG. 2.—*Torn capsule is tense in abduction, and the lips of the tear are drawn tightly together.*

No limitation of abduction,
No pain,
No chance of re-dislocation,
Much shorter after treatment
in consequence.

The following groups show the pernicious effects of fixation to the side:—

GROUP I.

Three patients, aged 17, 45, and 62, had no splints at all, but their arms were merely placed in slings. Two obtained perfect results, but the oldest had some osteo-arthritis, which rendered overhead action difficult. Even so, the duration of hospital treatment was only 24 days.

GROUP II.

Nine patients had their arms bandaged to their sides:—

Two for 7 days: (1) Æt. 59 years; 32 days' treatment;

result, perfect. (2) Æt. 69, 30 days' treatment; can get it overhead, but much osteo-arthritis.

One for 10 days: Æt. 53; 140 days' treatment; full movements, but took nine months to do it.

Three for 14 days: (1) Æt. 31; 30 days' treatment; marked pain and osteo-arthritis, abduction limited at 110°. (2) Æt. 37; 9 days' treatment; full work in three weeks. (3) Æt. 50; 84 days' treatment; after 18 weeks could only get arm up to right angle, and had bad osteo-arthritis.

One for 22 days: Æt. 60; 18 weeks' treatment; right angles only, still away from work.

Two for 28 days: (1) Æt. 56; full movements, but took 9½ weeks to get back even to light work. (2) Æt. 69; right angles only, after 17 months.

GROUP III.

Seven patients had had their arms abducted to a right angle on a splint for seven days:—

Æt. 3 years. Result, perfect.

Æt. 39. Arm can be raised easily above head. (This was a case of recurrent dislocation, the initial injury being some eight years ago; the man seems to be an epileptic; he did light work five to six weeks after reduction on the present occasion, and full work ever since.)

Æt. 41. Result, perfect. Full work after 54 days, and ever since.

Æt. 48. Can get arms above her head, but has osteo-arthritis in *all* joints. Can circumduct, slowly. Was discharged after 23 days, and has been at full work ever since. No medical treatment at home.

Æt 62. All movements full; constantly lifts 80–90 lbs. of fish with the affected arm. Discharged after 20 days' treatment, and has done full work ever since. He earns as much now as he did before the accident, lifting cases of fish on to his head and carrying them. No osteo-arthritis.

Æt. 67. Full movements; no pain; no osteo-arthritis; no loss of wage-earning capacity. Was discharged after 33 days. No further medical treatment at home.

Æt. 77. Circumflex paralysis (mentioned above). No limitation of passive abduction, and no osteo-arthritis.

By "perfect," it is meant that (i) the movements at the shoulder-joint are absolutely unimpaired; (ii) there is no pain; (iii) there is no loss of muscular power, or of wage-earning capacity. The cases in the second group (those who had their arms bound down to their sides) contrast very unfavourably with those in the third group; four of them can only lift their arms to a right angle or thereabouts, and all took an inordinately long time to achieve even this. On the whole, one may state that the longer they were tied down, the longer did they take to attain even a very mediocre result. Those in the last group, on the other hand, can *all* get their arms above their heads without pain or difficulty, except that one old man cannot do it voluntarily by reason of circumflex paralysis. The duration of treatment is very much shorter, and—another point of importance, though it is difficult to explain it—only one has since developed osteo-arthritis. Even this is a point of great importance; it would be worth while to adopt abduction as the standard method of treatment if only because the liability to post-traumatic osteo-arthritis is decreased thereby.

SUMMARY OF CONCLUSIONS.

1. The present after-treatment of dislocations of the shoulder by fixation of the arm to the side is irrational, unscientific, and, in actual practice, unsuccessful.
2. Limitation of abduction and osteo-arthritis are its results.
3. In rectangular abduction, the rent in the capsule is closely coapted, whereas in adduction, the capsule is crowded together in a crinkly lump, and coheres in that position, thus limiting abduction, and causing pain.
4. The arm is no more liable to re-dislocate when placed in rectangular abduction than when it is tied to the side.
5. If abduction is adopted, the resulting movements are much better; they are obtained much more quickly, and more painlessly.
6. These statements are based on fact, not on arguments.

Modern Conceptions of Heart Disease.

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OUR conception of various aspects of heart disease has undergone a considerable change during the last few years. The epoch-making work of Mackenzie, Lewis, and others, on the heart, largely by means of the electro-cardiograph, has thrown a flood of light on many problems hitherto unelucidated, and has led to a fundamental revision of many of our old views derived from the archaic teaching of the schools.

Mechanical View.—The old conception of valvular disease and its after-effects on the heart was largely a mechanical one. Infection was recognized as the initial cause of the valvular defect, but the after-results were explained mainly on mechanical grounds as due to the interference with the normal course of blood through the heart, leading to dilatation, hypertrophy, back pressure, and general venous engorgement; each chamber of the heart being affected in turn as its hæmostatics were interfered with.

It is true that mention is made in the text-books of infection involving the muscle of the heart, but this is rarely accentuated as the main factor; or, if present, it is assumed to have recovered with the healing of the valve lesion. Mechanical causes are assumed to be sufficient to explain heart failure; the heart muscle finally yielding to the long continued extra work it is called upon to perform.

Infective View.—The modern conception of valvular disease regards infection, or poisoning, of the heart muscle as the primary factor in the production of heart failure, and relegates to the mechanical factor a secondary and subsidiary part. A valve—the mitral, for example—may be attacked by the rheumatic or other infection; the infective process may stop and the valve be damaged, with a soundly healed scar leading to crumpling or puckering of the edges. Mitral regurgitation follows, but if the heart muscle has escaped damage by the infective process, the mere regurgitation of blood is insufficient to give rise to dilatation, hypertrophy, and so-called back-pressure effects. If the conducting and contractile muscle fibres are intact and healthy, such is the reserve power of the heart that it can maintain the circulation efficiently for all needs through a long and strenuous life, in spite of the permanent damage to the valve and the consequent regurgitation. Hypertrophy and dilatation, therefore, are in no sense a measure of, or in proportion to, the amount of regurgitation, but are rather an indication of the extent of the damage done to the cardiac muscle.

In support of this may be mentioned the following facts. It is known that (1) the experimental production, aseptically, of a valve lesion causing regurgitation is not necessarily followed by enlargement or by any change in the muscle; (2) cases of frank valvular disease may be found post-mortem to show no change in the muscle; (3) the largest hearts found post-mortem are frequently those in which there is no valve lesion discoverable, as in syphilis, renal disease, emphysema, adherent pericardium, and alcoholism.

Apical Systolic Bruits.—It follows, then, that a systolic murmur at the apex, even though it is due to a definite regurgitation from crumpling of the mitral valve, if unaccompanied by enlargement of the heart, and if the tolerance of the heart for exercise is good, may be neglected as of little or no moment as affecting the efficiency or life of the individual; a conclusion which has been abundantly corroborated by the observation of numerous soldiers suffering from such a lesion who have passed unaffected through prolonged periods of arduous strain.

The presence of an apical systolic bruit is, of course, by no means indicative of a lesion of the mitral valve. The certain diagnosis of early mitral disease is a matter of great difficulty. If there is a definite history of rheumatic infection, with a definite or even border line enlargement of the heart, the probability is strengthened that the mitral valve is structurally damaged. In the absence of such aids, the conclusion is by no means warranted. In at least 50 per cent. of cases definitely diagnosed as early mitral disease, the valve post-mortem will be found structurally intact. The cardio-respiratory bruit, the bruit of a lax mitral ring, and exo-cardial bruits give rise to confusion; they are sometimes capable of diagnosis and differentiation, but not infrequently cannot be distinguished, by the character of the bruit alone, from that of an organic lesion. The loudness and the character of the murmur are of no help; nor is the conduction of the bruit, for functional cardio-respiratory bruits may be equally well conducted through the axilla to the inter-scapular region.

Mitral Disease.—So far I have spoken only of early mitral disease, with a lesion limited to the valve edges causing crumpling and regurgitation, but with the heart muscle intact. Such a lesion does not necessarily lead to enlargement of the heart nor to impairment of its function. From such a comparatively simple early stage all degrees of valvular affection are found up to the more advanced condition of full-blown mitral stenosis, depending on the severity of the initial infection, or more commonly on repeated infection; or, as certainly happens more often than is fully recognized, upon long-continued chronic infection. It is obviously unscientific to speak of mitral regurgitation and mitral stenosis as though they were separate diseases; they are merely different degrees of the same process and should always be spoken of by the term "mitral disease"; for it is frequently impossible to say when one shades off into the other. For early mitral stenosis to develop, a period of at least three years is required after the original infection, and for a full-blown stenosis at least five years; during the whole of which time the chronic infective process has been going on. Hence the rarity of mitral stenosis in children under ten years of age. The important factor is, not the gradual stenosis of the orifice, but the concomitant infection and damage to the heart-muscle, which is proceeding at the same time; especially the damage to the conducting paths, the inter-ventricular node, and the bundle of His, where they lie in close relation to the mitral and tricuspid orifices. Here, again, the hæmostatic interference occasioned by the stenosis would not suffice to disable the heart, were it not that the muscle is weakened and diseased by the simultaneous infective process. Hence the supreme importance of the early diagnosis of mitral stenosis by the detection of a presystolic bruit.

The presence of a systolic bruit at the apex is no proof that mitral disease exists. The presence of a presystolic bruit is definite evidence that the valve is affected, and mitral stenosis connotes a continuous infection not only of the valves, but of the heart muscle also—a more or less generalized carditis.

It follows, then, that every case of recent infection showing a permanent as opposed to a transitory systolic bruit at the apex must be watched continuously over a period of at least three years before it can be safely stated that the mitral valve is not seriously affected, and a sound prognosis be given. The main importance of the bulk of systolic apical bruits lies not in their mere presence but the necessity they demand for careful observation for the earliest detection of a supervening presystolic murmur.

Aortic Disease.—Considerations analagous to those on mitral disease apply to aortic disease—with certain important modifications. Similarly, we should speak not of aortic regurgitation and aortic stenosis as two separate conditions, but of "aortic disease," the pathological process giving rise to both being the same. Regurgitation may occur without stenosis, but not stenosis without some degree of regurgitation. To diagnose stenosis it is necessary first to diagnose regurgitation.

The secondary changes in the heart of dilatation and hypertrophy are not primarily nor mainly due to the increased work called forth by the altered hæmostatics, but to the coincident carditis resulting from the infective process. It is common to meet with cases presenting an aortic leak, or even a decided degree of regurgitation—as judged by other signs than the diastolic murmur—which have carried on strenuous work for years without ill-effect. Cases in the army cropped up with considerable frequency and presented no symptoms, the defect being discovered accidentally in the course of routine examination for other purposes.

The risk of damage to the heart-muscle in aortic disease is accentuated by the proximity of the coronary arteries; if they become involved in the diseased process, the nutrition of the heart suffers, and inflammatory or degenerative changes become accelerated. Hence the greater seriousness, on the whole, of aortic over mitral disease.

To recapitulate: in the diagnosis of *organic disease of the heart*:—

(I) A systolic bruit alone is of no value.

(II) A systolic bruit with a permanent enlargement of the heart is definite evidence of organic disease; but it is the enlargement that matters, not the bruit.

(III) A diastolic bruit is definite evidence of an organic valve lesion; without a permanent enlargement, it is of relatively less import; with enlargement, there is definite evidence of carditis.

(IV) Enlargement, with or without a bruit, is definite evidence of organic disease.

Gradations of Infection.—All gradations of infection are met with in heart disease. A simple rheumatic infection may lead to slight crumpling of a valve and a soundly healed scar; the process becomes stayed, the heart muscle is intact, and the heart, for practical purposes, is little or no worse off than a normal heart. A further degree shows the same changes to a greater extent, more destruction and greater regurgitation, with involvement of the heart-muscle. The next degree is that of repeated acute infection or chronic continuous infection leading to stenosis and serious carditis. From this to the subacute bacterial endocarditis of Libman is but a short step; further degrees merge imperceptibly into the more advanced condition of acute infective endocarditis up to the most malignant forms of this fatal disease.

Compensation.—The crippling factor in valvular disease is not the mechanical interference with the function of the valve, but the invariable coincident infection of the heart muscle. The greater the destruction of the valve the greater the degree of infection of the heart muscle and the more liability to dilatation, hypertrophy, and ultimate heart failure.

It follows, then, that the old mechanical conception of compensation in heart disease is no longer tenable —that the functional efficiency of the heart depends upon a nice adjustment between a crippling dilatation and a compensatory hypertrophy; and when the former is in excess of the latter, compensation is broken and heart-failure sets in. The product of armchair reasoning, based on *à priori* grounds and unsupported *à posteriori*, it may now be finally discarded as an inadequate explanation of the mechanism of heart-failure.

Chronic Infection.—The conception that the heart may be subject to low forms of chronic infection (other than the rheumatic infection), though not new, has certainly assumed greater prominence in recent years. Organisms of various kinds have been found in the heart muscle; the difficulty is to diagnose their presence during life. It is not unlikely that some of the intractable cases of "D.A.H." following infection—dysentery, trench fever, etc.—will ultimately be found to be due to definite cardiac infection. Major Byam has recently shown that the blood in trench fever may remain infective for as long as 443 days; so that many cases of D.A.H., thought to be due to the after-effects, may still be suffering from the disease.

Damage to the heart muscle by chemical poisons such as alcohol and tobacco, or by bacterial toxins as in diphtheria, is well recognized. Clinically, it is often impossible to distinguish between infection and intoxication.

The *practical point* is that all cases of infection should be carefully watched with respect to heart trouble; that the period of convalescence from acute infective disease should be lengthened beyond that commonly thought necessary; and that all cases of apparently unimportant heart lesions should be carefully shielded against infection.

Functional Efficiency.—As the outcome of the foregoing considerations our outlook on valvular heart disease has undergone material change—from the prognostic point of view. No longer are we obsessed by the importance of murmurs when unaccompanied by other and more important physical signs. It has become recognized that the functional capacity of the heart to perform its appointed task is of more importance than the mere structural defects. If the exercise-reaction of the heart is good, the tolerance of sustained exercise equally good, and there is no enlargement of the heart, there is strong presumption that the muscle is undamaged; and systolic murmurs of whatever origin may safely be neglected as of little or no moment. If a diastolic bruit is present, either at the mitral or aortic area, there is certain evidence of structural organic disease; but here, again, if the exercise-reaction and tolerance are good, and there is no enlargement, a good prognosis may be given. More careful watching is required, however, of the future progress of such cases, for there is more likelihood of muscle damage having taken place, or of slow chronic infection going on.

Rhythm of the Heart.—Perhaps the most striking change in our outlook on cardiac affections lies in the domain of abnormalities of rhythm.

The chief forms of irregularity of which we have now a more or less exact knowledge are—

(1) *Sinus Irregularity of the young*, in which the stimulus begins at the normal place, the sino-auricular node, and proceeds along the normal course, evoking a normal contraction, but occurs at irregular intervals. It is a disturbance of the pacemaker of the heart, and is probably a vagus effect. Respiratory arhythmia is a typical sinus irregularity. So far from being an indication of disease it is rather, as

Sir James Mackenzie points out, presumptive evidence that the heart muscle is healthy, from the fact that response is so ready to vagus variations. Formerly young subjects presenting sinus irregularity were needlessly "treated" or kept at rest for prolonged periods from a mistaken notion of the seriousness of the condition.

(2) *Sino-auricular Block.*—A relatively uncommon form in which the stimulus arises at the sino-auricular node and pursues a normal course, but every now and then fails to materialize, and a pause occurs in which the whole heart is at rest. Clinically, it cannot be distinguished from other varieties of heart-block without the aid of the polygraph or the electro-cardiograph.

(3) *Extra-systoles.*—Irregularities of rhythm due to extra-systoles are extremely common and usually of little moment. The stimulus to contraction may arise in an ectopic irritable focus anywhere in the musculature of the auricle or ventricle, or in the tracts of conducting tissue. They are recognizable by auscultation and palpation, but to determine their origin instrumental aid is required. The polygraph will in most cases show whether they are auricular or ventricular; by the electro-cardiograph may be determined the exact site of origin, that is, in which region of auricle or ventricle or of nodal tissue they arise. Usually they are most in evidence when the pulse-rate is slow, and tend to disappear if the heart is accelerated by exercise or emotion, when the rhythm becomes regular. Extra-systoles that appear only after exertion, but are absent when the heart's action is slow and quiet, are of more serious import, and may indicate some mechanical interference with contraction.

Extra-systoles may be present all through life without impairing the efficiency of the heart. They may exist for long periods at a time, and then disappear to be replaced by a normal rhythm. They may appear temporarily as the result of toxic or infective influences or of drugs. Usually regarded as of little moment they cannot altogether be ignored, for granted that they may not interfere with functional efficiency they, at any rate, are the expression of an undue irritability of the heart and are in this sense abnormal.

(4) *Paroxysmal Tachycardia,* in which the whole heart beats in orderly sequence at a greatly enhanced rate. The stimulus to contraction arises not in the normal place, the sino-auricular node, but in some ectopic focus in the wall of the auricle. The characteristics of paroxysmal tachycardia are the sudden onset, the absence of variability in rate to changes of posture or to exercise, and the equally sudden offset after a variable duration of hours or days. Each auricular impulse evokes a corresponding ventricular contraction and the pulse is regular throughout.

Paroxysmal tachycardia shades off imperceptibly into the still more violent disturbance of

(5) *Auricular Flutter,* in which the auricle, in response to stimuli from an ectopic focus, beats at a greater rate still—up to 300 a minute. The line between P.T. and auricular flutter is arbitrarily fixed at about 200 per minute. Up to and somewhat above that rate the ventricle can respond to each contraction of the auricle and a pulse-rate may appear of 240–50 per minute, auricle and ventricle contracting sequentially. Beyond this rate the ventricle cannot go the pace, and a condition of heart block appears—usually a 2–1 block—in which the auricle may be contracting at 300 a minute but the ventricle only at 150—or, with a 3–1 block, at 100. Clinically, auricular flutter can be recognized if the case is watched carefully, but it is impossible to measure

the rate of auricular contraction except by means of the electro-cardiograph.

The condition occurs mainly in middle or late life and usually appears intermittently at first, but tends to become established and to endure for months or years. Vigorous administration of digitalis may convert the flutter into auricular fibrillation, when the pulse assumes the characteristic irregularity of this condition. On withdrawing the drug the heart may suddenly settle down to a normal rhythm.

(6) *Auricular Fibrillation,* which gives rise to the common irregularity of a failing heart, when the pulse is wholly irregular, no two successive beats being alike in time and force. The auricle has ceased to contract as a chamber, the contraction being replaced by tremulous flickering of individual fibres or bundles of fibres in response to minute ectopic stimuli. The ventricle responds irregularly to such stimuli as are strong enough to get through the A.V. node and the bundle of His, and the characteristic irregular pulse appears. Those physical signs disappear, which are dependent upon the auricular contraction; a presystolic bruit, the auricular wave in the veins of the neck.

Auricular fibrillation is usually recognized clinically with ease and without instrumental aid. Occasionally, tumultuous extra-systoles may lead to confusion, in which case a simple pulse-tracing is generally sufficient to enable a distinction to be made. The polygraph will at once show the absence of the A wave in the venous tracing, and may show the minute waves due to fibrillary contraction. The electro-cardiograph will bring out these waves, and show the absence of the P wave of the auricular complex, thus rendering the diagnosis certain.

Fibrillation may occur intermittently at first, but usually, once firmly established, it endures for the rest of life. Occurring in a heart the subject of the rheumatic infection, it is the condition par excellence that is definitely benefited by digitalis.

A new conception is that of *ventricular fibrillation;* that, in the terminal stage of heart-failure, the end may be due to the onset of fibrillation of the ventricles with resulting failure of contraction and inevitable death. Further, that some cases of sudden death may be due to fibrillation of the ventricles without necessarily an antecendent auricular fibrillation. For example, a sudden obliteration of a coronary artery may set up ventricular fibrillation.

(7) *Heart-block.*—The condition known as heart-block, in which the stimulus from the auricle fails to reach the ventricle owing to a block in the conducting paths, is recognizable clinically without instrumental aid. A pause occurs of varying duration during which no heart sound is heard. Occasionally, it is claimed, the auricular beat may be heard, but this is rare. To distinguish heart-block from sino-auricular block, or to determine the site of block, graphic records are required. The polygraph will show the auricular waves in the venous curve and enable the ratio of auricular to ventricular beats to be determined; that is, the degree of heart-block, whether 2–1, 3–1, and so on; or will show if the dissociation between auricle and ventricle is complete, in which case complete heart-block is present, when the ventricle takes on its own idiopathic rhythm of about 30 to 40 beats per minute, the auricle contracting at the normal rate. From a persistent abnormally slow pulse-rate, heart-block may usually be inferred without the necessity of taking tracings. The electrocardiograph will demonstrate the presence of heart-block with far greater certainty than the polygraph, and may indicate the site of block, whether in the A–V node, or the bundle of His, or in the right or left

branch of the bundle—the so-called right or left bundle block.

(8) *Pulsus Alternans.*—A pulse curve of regularly alternating strong and weak beats, regularly spaced in time, is known as *pulsus alternans* and is usually held to indicate a failing myocardium. Seldom can it be recognized with certainty by the touch alone; a sphygmographic tracing is necessary to determine its presence. The ordinary Dudgeon sphygmograph is far superior to the polygraph for this purpose, the radial tracing obtained from the latter being frequently not sensitive enough to show the alternations. Sometimes *pulsus alternans* may be heard when taking the blood-pressure by the auditory method; or it may be seen when the Pachon oscillometer is used. The electro-cardiograph may give no indication of *pulsus alternans.* A case may furnish a polygram showing well marked alternations and yet in the electro-cardiogram the R and T waves of the ventricular complex may be of uniform height throughout; or may show alternations inversely to those of the polygram. The electro-cardiograph gives a measure not of the force of auricular and ventricular contraction, but of the amount of electrical disturbance evoked by the stimulus to contraction.

It is a wise precaution to take a sphygmographic tracing of all cases of feeble pulse, especially of the senile heart; for a *pulsus alternans* will often indicate impending danger when other signs may not suggest its imminence.

To sum up: the unravelling of irregularities of rhythm and the establishment of their diagnosis and relative import on a sound basis are an immense advance in our knowledge of heart disease; they have replaced the old nebulous notions by a new and clear conception on which treatment and prognosis can be based with some degree of precision.

Recent Work on Tropical Diseases.

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MALARIA.—Stephens and co-workers[1] have studied the action of various drugs and preparations of quinine in malaria. They find that novarsenobillon in doses of 0·9 gram intravenously causes the disappearance of the benign tertian parasite in all its stages from the peripheral blood within 24 hours of administration, its action being even more rapid and efficient than quinine. On the other hand, this drug exerts no influence in cases of infection with the quartan and sub-tertian parasites. Three groups of cases were treated as follows :—

> *Group* 1.—A single intravenous injection of novarsenobillon, 0·9 gram, on the first day with an intramuscular injection of quinine bihydrochloride, grains 15, on the first and second days of treatment (41 cases).
>
> *Group* 2.—An intravenous injection of novarsenobillon, 0·9 gram, on the first, eighth, and fifteenth days, with intramuscular injections of quinine bihydrochloride, grains 15, on the first, second, eighth, ninth, fifteenth, and sixteenth, days of treatment (12 cases).
>
> *Group* 3 (control).—An injection of quinine bihydrochloride, grains 15, on each of two consecutive days only.

All the cases were infectious with the benign tertian parasite.

Of group 1, 36 per cent. of the cases relapsed within 65 days; of group 2, 17 per cent. of the cases relapsed, and of group 3 about 80 per cent. of the cases relapsed.

Stephens and co-workers[2] also tested quitinene hydrochloride, an oxidation product of quinine, on benign tertian malaria and found it of no value.

Bass[3] calls attention to the treatment of malaria which has been adopted after a study of different methods in more than 25,000 cases. The treatment for adults is 10 grains of quinine sulphate every night before retiring for a period of *eight weeks.* For children the doses that give the same results as 10 grains in adults are: under one year, ½ grain; one year, 1 grain; two years, 2 grains; three and four years, 3 grains; five, six, and seven years, 4 grains; eight, nine, and ten years, 6 grains; eleven, twelve, thirteen, and fourteen years, 8 grains; fifteen years or older, 10 grains. The 6, 8, and 10 grain doses are best administered in the form of two tablets, the smaller doses in some aromatic syrup. Persons who have acute attacks should be given one dose of 10 grains (or a proportionate dose for children) three times a day for three or four days, which always relieves the acute symptoms, and then the eight weeks' treatment described. Bass claims that this treatment will disinfect more than 90 per cent. of cases, but it must be rigorously adhered to, and some persons require longer treatment than others. He remarks that hypodermic inoculation of quinine is very unpleasant, and should be reserved for pernicious malaria.

In view of the frequent administration of quinine intravenously the studies of McCarrison and Cornwall[4] on the "pharmaco-dynamics" of quinine salts are of considerable interest and importance. They find that all the usual salts of quinine employed for intravenous medication are dangerous to life in large doses. The respiratory centre is more seriously affected than the cardiac centre. All salts of quinine cause a profound fall in blood-pressure not accompanied by a cessation, or even a diminution, in the strength of the heart-beat. The fall of blood-pressure is usually recovered from in four or five minutes, but the period of cardiovascular depression may last for a considerable time. The dilution of the quinine with a large volume of salt solution or with a gum-arabic solution does not compensate for its depressor action. Intravenous injections of quinine should be given very slowly, and should be administered with great caution when the general condition of the patient is bad, and when the blood-pressure is low. Adrenalin given with the quinine counteracts to some extent the immediate and dangerous fall of blood-pressure which may result from quinine alone. McCarrison and Cornwall consider that the intravenous injection of quinine should be reserved for cases of special urgency, that where possible the hydrobromide in doses not exceeding 15 grains should be used and the injection combined with not more than 0.3 cc. of the commercial solution of adrenalin in all cases in which the blood-pressure is below 100 mm. of mercury. The quinine injections should be given only when the parasites—as indicated by blood examination—are susceptible to its action, viz., when in the stage of sporulation.

Lipkin[5] has studied the distribution and destruction of quinine in animal tissues. Evidence is given which suggests that areas exist in the blood-vascular system which remain almost free from quinine during a period of quinine medication—an important fact which may explain the inefficiency of quinine to cure malaria, *i.e.*, to destroy all parasites. Quinine accumulates in most tissues in much higher concentration than in the blood, the adrenals being pre-eminent in this respect. A quinine-destroying agent, an enzyme, can be extracted from the liver.

An excellent account is given by Dudgeon[6] of the pathological changes in fatal cases of pernicious malaria caused by the sub-tertian parasite. Of comatose cases, one group showed the cerebral capillaries engorged with numerous infected red-blood cells, free parasites, melanic particles, and prominent and detached endothelial cells; another group showed punctiform hæmorrhages in connection with the small blood-vessels, without massing of parasites in the cerebral vessels, referable to the general toxæmia of the infection.

The heart-muscle showed fatty degeneration in 23 out of 45 cases examined. The adrenals were frequently involved, particularly in cases showing muscular weakness, low blood-pressure, and diarrhœa and vomiting. The chief change in these organs was a reduction in the fatty lipoids of the cortical layers, in a few cases thrombosis of capillaries with hæmorrhages were observed. The intestines frequently showed congestion of the blood vessels, with occasionally hæmorrhages into the villi and submucosa.

Gordon Thomson[7] has shown that complement fixation tests can be carried out in malaria by means of an antigen prepared with cultures of the malaria parasite—for full details the original paper must be consulted. The experiments suggest the possibility of a specific reaction for benign and for malignant tertian malaria as well as a group reaction. This indicates that it is probably better to employ as antigen one made from a mixture of cultures of the various parasites. Positive reactions are also obtained with syphilitic using the malaria antigen, so that it is necessary to exclude this disease in complement fixation tests for malaria.

Cholera.—In *Nature* for June 5, 1919, Prof. Bayliss advocated that gum-arabic solution, which gives such good results in shock, should be tried in cholera in order to obtain a more prolonged maintenance of the blood-pressure than sometimes follows the use of hypertonic salt solution. Sir Leonard Rogers,[8] therefore, made a trial of gum solution, but with disappointing results, several cases being lost which might have been expected to recover with hypertonic salt solution. Moreover, instead of the great relief, often resulting in sleep, before the hypertonic saline injection is finished, the gum solutions were followed by increasing distress, such as difficulty in breathing and cyanosis. Lt.-Col. A. Leventon, I.M.S., has also extensively tried gum solutions in cholera—in over 1,000 cases—with results in entire accordance with those of Sir Leonard Rogers.

Prof. Bayliss, in commenting on these disappointing results, suggests that they may depend on retention of toxins, which would probably be favoured by the gum solution, the object of which is to retard passage of fluid out of the vascular system and so maintaining the blood-pressure. He is more inclined to think, however, that the symptoms mentioned are due to some mechanical action of the gum solution.

Kuhne[9] describes an interesting method of treatment of cholera. It consists in the use of drinks of kaolin in water—100 grams of kaolin in 250 cc. of water. Of this a glass (about 90 cc.) is given every ½-1 hour. It is rarely necessary to give more than six glasses in the first 12 hours; generally the vomiting soon ceases, the pulse improves, and the patient sleeps. During the second 12 hours and the following day, several glasses of the mixture are given according to the patient's condition, together with light diet for a few days. For the first 18 hours from the beginning of treatment, except for water, neither food nor drink should be given.

Dysentery.—In cases of amœbic infection, MacAdam[10] advocates treatment by a course of 18 grains of emetine, spread over 12 days, 1 grain being given hypodermically and ½ grain orally each day. He claims that the results of this treatment show a considerable improvement on those obtained after a course of emetine by injection alone and compare favourably with the results obtained with full courses of emetine bismuth iodide.

Brug[11] has investigated the nature of *Iodine cysts*, first mentioned by Wenyon. They are cysts not unlike tetragena cysts, but when treated with iodine show a deeply-staining corpuscle, the nucleus rarely becoming visible. Brug considers that the iodine cyst is the cystic stage of an amœba, *Entamœba Williamsi.*

Leishmaniasis. — Connor and Shortt[12] discuss *Leishmania tropica* infections in Mesopotamia. As regards treatment, they suggest removal of crusts and hot fomentations for cleaning. If much heaped up, CO_2 snow is the best caustic, after which methylene blue (3 per cent.), moist eusol dressings, or strong iodine may be applied, alternating with hot fomentations, to promote healing. When the ulcer is clean and healing, zinc sulphate ointment may be used, or it may be covered with ambrine.

Acton[13] has studied the distribution on the body of Bagdad boils as a key to the nature of the transmitting agent. They are found only on exposed parts of the body, which suggests some blood-sucking dipteron and not blood-sucking insects, such as the bug, flea, or louse. The boils chiefly occur on thin hairless areas of skin, suggesting that the proboscis of the blood-sucking dipteron is small and not very penetrating. He suggests that the anatomical distribution of Bagdad boil corresponds more closely with sandfly bites than with the bites of any other blood-sucking diptera.

Yaws.—While salvarsan and neosalvarsan are, without doubt, the specific remedies for yaws, scarcity of these drugs and their high price led Castellani in 1915 to advocate an antimony mixture containing also sodium salicylate and potassium iodide as a remedy in place of these drugs. The formula is—

Tartar emetic	0·065 gram.
Sodium salicylate	0·65
Potassium iodide	4·0 ,
Sodium bicarbonate	1·0 ,
Water	30·0 cc.

This forms one dose, which is diluted to four ounces (120 cc.) with water and given thrice daily to adults.

Guerrero, Domingo and Arguelles[14] have used this treatment in 43 cases in the Philippines, where the disease is prevalent. Seven of the cases discontinued the treatment early, and of the remaining 36 cases, 24 were completely cured, seven improved, and five showed no improvement. They remark that the treatment of Castellani undoubtedly exerts a curative influence on the various manifestations of framboesia. In six or seven days of treatment the granuloma becomes livid in appearance and surrounded by a pinkish halo, and begins to flatten. The crust dries and gradually disappears, leaving a macule that eventually disappears. In deep and extensive ulcers cicatrization takes place gradually and concentrically, the process lasting 15–30 days. Pain in the bones, pain and swelling of the joints, and pruritus are relieved early in the treatment, although the pain in the bones and joints sometimes recurs even after complete healing of the lesions.

If the mixture causes emesis and iodism, they may be prevented by increasing the sodium bicarbonate, or by giving 15 minutes before each dose, a dose of paregoric or of codeine.

Bilharziosis. — Christopherson[15] has re-introduced antimony treatment for both the vesical and rectal forms of this disease. The solution used consists of antimony tartrate, gr. ½ in 20 minims of distilled water. This is diluted again before injection with once or twice its volume of saline solution. The drug is injected intravenously into an arm-vein, care being taken that the whole injection passes into the vein, otherwise sloughing may occur. Injections are given at first every day and subsequently every other day, the dose being increased by gr. ½ of antimony tartrate at each injection until gr. 2 or gr. 2½ are reached, the total quantity of antimony tartrate forming a course being 20–30 grains. In the vesical form the urine is microscoped every day. For this purpose the urine is collected in a conical glass and allowed to sediment for at least half-an-hour. The sediment is then taken up with a pipette and examined microscopically. When the ova are numerous, a drop or so on a slide suffices, but as they become scanty, as much as 5 cc. of the sediment are placed in a shallow glass capsule. In the rectal form, a saline purge is given and the stool examined on a slide. If no ova are found, the whole stool is poured into a glass jar and diluted with about three times its volume of water, to which 1 oz. of formalin (40 per cent.) is added. The mixture is allowed to stand for half-an-hour and the supernatant fluid poured off. This process of washing is repeated 4 to 6 times until the fluid is clear. Slides are then prepared from the residue and examined.

The injections of antimony are given with care, the patient lying down, and preferably the first thing in the morning.

The first noticeable change with treatment is the disappearance of visible blood, a mere smokiness perhaps remaining until 14 days after about 20 gr. of antimony have been given; then the urine becomes quite clear and normal.

Christopherson maintains that the drug kills both the ova and the adult worms. The parent worms are probably generally killed after a course of treatment with 25 gr. of antimony tartrate, but living ova may continue to be discharged for some time, up to four months after this event, but gradually become less and less in number and ultimately disappear. Occasionally a true relapse may occur, in which case the course of antimony treatment may be repeated. The symptoms are due almost entirely to the irritant effects of the *ova*, the parent worms, which inhabit the portal vein and its tributaries, causing no symptoms except certain microscopic blood-changes with some anæmia and sallowness.

N. Hamilton Fairley[16] has investigated the immunity reactions in Egyptian Bilharziasis. He finds that following invasion by bilharzia cercariæ, at about the fifth to eighth week of the disease, a leucocytosis of 34,000, together with an eosinophilia of 64 per cent., may be present. After persisting for some weeks, a steady decline follows for some months, so that from the sixth to the eighteenth month the leucocytosis will be about 10,000 and the eosinophilia 13 per cent.

He has also devised a complement fixation test. The antigen is prepared from the liver of snails (*Planorbis boissyi*) infected with bilharzia—either a saline or an alcoholic extract. This gives definite complement fixation with the blood-serum of infected persons.

Ankylostomiasis.—Wrench[17] gives the results of various methods of treatment in this disease. Manson's mixture—chloroform ♏ 45, eucalyptus ♏ 30, and castor oil ʒ 10, given in two doses with half an hour interval before the morning meal—failed to cure a single one out of 19 cases, the treatment being given six times.

Thymol, gr. 60, preceded and followed by a saline purge, cured four out of 12 cases—one by 3, one by 4, and two by 5 treatments.

Chenopodium oil proved the best remedy, curing 40 out of 45 cases—ten by 1, ten by 2, seven by 3, seven by 4, five by 5, and one by 6 treatments.

The treatment is as follows: at 1 p.m., 2 p.m., and 3 p.m. one capsule containing 1 cc. of chenopodium oil, and at 4.30 p.m. 6 drachms of castor oil. The oil is toxic and must be used with caution, for it may cause vomiting, giddiness, and even slight collapse.

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Observations on the Cause and Treatment of Abdominal Hernia.

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DURING the last five years I have been in medical charge of a German prisoners-of-war camp, and have kept notes of the men passing through. The total is about 27,635, and all were naval and military prisoners, except the first 1,511, who were "civilians" taken in this country or on board enemy ships, many of them being German reserve officers and men endeavouring to return home to join the colours.

A large number of men had a pendulous abdomen, resembling in size the appearance of a fifth-month pregnancy; many were suffering from bubonocele or hernia. In young Germans, this condition of the abdomen is due to the lack of "sporting" exercise usual with young Englishmen, and to the "sloppy" character of their food, especially the liberal potations of "small beer."

In the following I exclude all ventral herniæ, caused by either G.S.W. or appendicectomy operations.

I have no means of obtaining a record of the percentage of hernia in the British Army, or the percentage of rejections amongst volunteers or conscripts. Many men have been sent home from France suffering from hernia, and have been admitted to the auxiliary hospitals under my charge, but this is no evidence of a general condition.

The 1,511 civilian prisoners showed	3·55	per cent.	of hernia.
The 1st 10,000 soldiers and sailors	2·33	„	„
The 2nd 10,000 „ „	2·53	„	„
The remainder - - -	2·36	„	„

The last 20,000 men had an average age of 20 to 25, and were not physically A 1, being of poor condition, with deficient teeth, and many had an ill-developed mentality, whereas the first 10,000 fighting men were generally a fine body.

The natural admiration of the German for something big tends to make him think that gross bulk is something to be admired; the average British soldier considers "fatty" to be an object of ridicule, and his condition as undesirable.

The explanation given by many, that they had to perform manual labour for which they were unaccustomed, truthfully explains the causation of their hernia, and is a strong argument in favour of the preventive measures I am suggesting.

The medical practitioner knows there is a line of demarcation between the normal and abnormal amount of fat in the individual, but he has difficulty in fixing the point at which the former ends and the latter begins.

A pendulous abdomen, or a pendulous condition of the tissues of face and neck, is not caused by fat. Abnormal fat causes rotundity, and does not give rise to the appearance of flaccidity. Flaccidity is a symptom due to the loss of contractile power of muscles. When there is fatty infiltration of muscles, it does not give a pendulous appearance until there is re-absorption of fat, as in wasting diseases.

This loss of muscular elasticity has the same effect in the young and the old. Premature age may attack any part of the human frame, not necessarily the whole; the skin, blood-vessels, or muscles may be the point of selection, and I believe it is always a symptom of a pathological change in the central or peripheral nervous system affecting a special anatomical area, in describing which we speak of part of the result, and not the cause.

For instance, a hernia may be a symptom of a lesion of the spinal cord causing local paresis, or again, alarming intestinal distension may be secondary to extensive bruising of muscles and nerves of the abdominal wall, a condition not infrequently seen in hunting counties. I have not seen a surgical operation cure either condition.

If the flaccid muscle is due to nerve exhaustion, we call it functional, if due to pathological change in nerve or muscle cells, we give it the name of a special disease. The one is temporary, and the other a permanent condition.

Hernia may have a functional or pathological cause, and the muscles concerned may be able to resume duty or be permanently incapacitated. It may be *symptomatic* or *accidental.*

The anatomical explanation is simple. There is always a pouch of peritoneum in the normal openings of the abdomen, and the arrangement of muscles round these openings with their nerve supply supports the theory of their conjoint sphincter action.

For example, the transversalis and external and internal oblique have equal action, if this is so, they will temporarily close the internal ring; in proof of this, in the upright position, watch the enlargement of the veins in the spermatic cord, when the abdominal muscles are forcibly contracted. If these muscles become incompetent, pressure from within causes the pouch of peritoneum to become longer, and thus makes a way for the gut to descend, producing the *symptomatic* form of hernia.

The comparative immunity of animals from hernia is in a great measure due to the fact that they do not assume the erect position. I am unable to obtain exact authority, but understand that the disability is not uncommon in monkeys.

The sudden or *accidental* cases of hernia are caused by these sphincter muscles being taken unawares, and when they regain their temporary loss of power, produce the phenomena of "strangulated hernia." Strangulation is most often found in recent cases, not in those of long standing; in old cases, it is probably due to the gradually dilating ring arriving at a "live" muscle area.

A scrotal hernia may contain several feet of gut with many adhesions, but no symptoms beyond great inconvenience. Recently, a case of left scrotal hernia was sent to me for operation, which contained 23 inches of large intestine loaded with fæces. Without enlarging the ring, reduction of the gut with its contents was impossible. The only symptom was that it felt "so heavy"; there was neither vomiting, pain, nor physical signs of constricted gut. The patient was a decrepit man of 62, and the explanation is that the sphincter of his inguinal ring had permanently gone out of action, and the muscles and gut were tolerating each other.

I believe there is sphincter control of the inguinal canal, Heselbach's and Petit's triangles, the umbilicus, obturator foramen, and the various openings in the diaphragm.

Congenital and acquired herniæ are due to incompetent muscular action, the intra-abdominal pressure being greater than the resisting power of the extra-abdominal structures. The absence of the power of resistance is caused by (*a*) an anatomical deficiency (as in congenital hernia); (*b*) loss of nerve power from disease or senile change; (*c*) deficient muscular control, either from lack of physiological use, or secondary to nerve disability.

The subjects of hernia are not confined to the class employed in manual labour, for 75 per cent. of cases arise in the adolescent and those employed in sedentary occupations; in other words, those who have not had time to develop (the very young), and those who have not kept in working order the whole of their muscular organization. The remaining 25 per cent. of cases are due to pathological change.

Take the attitude of a clerk at his desk, the average man of 40 lets his abdomen " go," and does not keep his abdominal muscles in a condition of semi-contraction, they are always relaxed; if placed on his back on the floor, he is unable to rise without the use of his hands, showing he has muscular incompetency.

A man of sedentary life plays violent tennis or football one day a week; during the other six days his muscles have been lying fallow, and he suddenly gives them work for which they have not been trained, so that they are not up to it. By the fixation of his diaphragm, the intra-abdominal pressure is easily greater than the external abdominal resistance; the muscle gives, perhaps, slightly in the first instance, but in time makes way for the greater power, and the surgeon is consulted for a bubonocele.

The abdomen of this class of man can be palpated to the spinal column, for there is no real area of resistance; his abdomen resembles a rubber bag which has lost contractile power, and passively submits to inflation, its contents remain only after the pressure has become equal within and without.

Very fat persons seldom develop hernia, the probable explanation being that massive blocks of adipose tissue in the omentum find exit difficult and the fatty infiltration of the mesentery anchors the gut.

In the upright position, the line of abdominal health is a gentle slope downwards, commencing at the Ziphoid cartilage to a point one inch in front of the pubes; the converse of this denotes the physically unfit.

Hernia is of more frequent occurrence in the ill-developed than in the athlete or the corpulent.

The symptoms of commencing inguinal hernia were briefly described by me in THE PRACTITIONER of February, 1918; this is a repetition of my summary :—

"The following symptoms are complained of, and I have seldom heard of any feeling of discomfort *below* the level of the umbilicus. In their order of frequency these are :—An uneasy feeling in the epigastrium but not actual pain, discomfort of a 'dragging' character referred to one or other costal margin, or its immediate neighbourhood, pain in the back, corresponding to the ninth and tenth ribs, a feeling of nausea, but no vomiting, and often constipation."

The recognized systems of surgery do not describe the physical signs preceding the actual bubonocele; the symptoms often continue definitely for months, and my colleagues agree with my observations concerning this condition.

On more than one occasion, I have seen the application of a truss give relief in 24 hours in men who had been taking large quantities of bismuth for a considerable number of weeks, with belladonna plasters applied "fore and aft."

We fail to recognize the symptomatic hints, and wait until the gross lesion has made its appearance. The general practitioner sees these early cases, the physician sometimes, the surgeon never. "Wait and see" may be a political axiom, but in surgery it is usually ignorance on the road to a catastrophe.

Preventive treatment resolves itself into keeping the muscles of the abdomen in a state of efficiency by daily drill in the habit of keeping the rectus and oblique muscles in a slight degree of tension; healthy people should exercise on the floor, sitting up and reclining alternately without using the arms. The passive movement of the abdomen during normal respiration is not sufficient to keep the muscles "fit." Deep breathing exercises are invaluable, especially in children.

The application of trusses is frequently made in a crude way; a measurement is taken, and the word "inguinal" or "femoral" is added, but the order given does not record the amount of pressure required, which should be stated in ozs. or lbs. The average commercial instrument gives not less than 3½ lbs. to 5 lbs. pressure, which is much in excess of the usual requirement and causes a great amount of harm.

In an early case, the pressure of a truss should be transverse, outwards, and slightly upwards; the ordinary instrument exerts pressure upwards and inwards, and in no way governs or controls the internal ring. In my opinion, the pad should be wedge-shaped, with the lower edge on a level with the anterior superior spine, and the base over the internal and not over the external ring; this suggestion is an inversion of the pad now on trusses.

Pressure should be accurately measured by a spring balance and pelvimeter, and the instrument-maker instructed to supply a spring with a compression of so many ounces, or possibly pounds; unless this is definitely stated, the muscles will atrophy from too much pressure, or the intestine will come down from too little resistance.

Operative treatment.—A general survey of the patient is necessary, for it is not a mere matter of sewing up a gap; the galvanic reaction of his abdominal muscles must be investigated, and age considered an all important item in the matter of repair.

"Winter cough," physical deterioration, renal insufficiency, or cardiac debility, negative operation.

Children do well, the causation being different in their case, for it is usually the outcome of an anatomical error.

In the various operations recommended it seems to be forgotten that the muscles have failed, and are in such a condition that they are unable to act as a permanent block to the descent of intestine. However carefully a coat is darned, if the material has "perished" it will give way again in the line of the stitches; so, too, with anatomical material, inefficient muscles will fray out, or scar tissue will spread, and, after two years or less, a surgical failure will be exhibited.

True resistance may be obtained by producing adherence of fascia to fascia; stitching the aponeurosis of the external oblique to the pelvic fascia gives an excellent wall, especially if a layer has been rolled before fixing, or turning in the aponeurosis under Poupart's ligament will be found satisfactory. Bassini's operation depends upon the muscles remaining in action, but practically 75 per cent. do not. Silver-wire filigree is a help but not a cure. Umbilical hernia is often cured by crossing half of each rectus muscle and fascia.

Briefly, muscular resistance cannot be depended on and a more stable material must be found; the nerve supply should be minutely demonstrated at an operation, and guarded and retained as far as possible in its normal position.

The Ciliæ of Respiration.

By C. O. JONES, M.Sc., M.R.C.S., L.R.C.P.
Late Capt. R.A.M.C.

THE functions of the ciliæ of the respiratory tract have not been studied nor given the importance which is their due. It is recognized that, by their rhythmical waving action, they move along mucus and foreign bodies towards the external world, but that cannot be their only function; their movements must influence respiration and be influenced by the respiratory movements, and disorder of the normal ciliary movements must produce symptoms and have an effect on the individual.

In the frog and lower animals, ciliæ are more widely spread than in man. The reason for their disappearance in certain places in the respiratory tract in man is the erect position. In the frog, the nostrils and lungs approximate to a straight line; the air taken in during inspiration in its course from the external world to the lungs impinges on the walls, and there deposits foreign bodies and bacteriæ which adhere to the sticky surfaces of the ciliæ, so that, in order that the air should be filtered, a long column of ciliæ is necessary. In man, the intake of air through the nostrils takes on more or less of a right angle with the respiratory tract, so that the air taken in at the nostrils is filtered by the walls and also impinges against the posterior wall. Thus, in the naso-pharynx impurities are filtered out of the air, and are waved outwards to the external world. By keeping the bacteriæ on the move, they are prevented from doing any damage.

The irritation of the ciliæ by foreign bodies, and the resulting hindrance to their rhythmical movement, causes the outpouring of mucus. This has probably some bactericidal action; secondly, it helps to float away the invader, and it dilutes and neutralizes the toxins, as will be seen later.

If the bacteriæ are allowed to remain for any length of time in one position, owing to diminished movement or destruction of ciliæ, toxins are poured out which paralyse or destroy the ciliæ, mucus cannot be poured out by the epithelium, the bacteriæ get a footing, and inflammation results.

Such, then, is the action in a normal animal in normal conditions. Extremes of cold and heat affect the movements of ciliæ. A cold draught will slow down their movements and, if continued, it will enable bacteriæ to obtain a footing; if this occurs, the toxins produced will put the ciliæ out of action, and nasal or naso-pharyngeal catarrh will result. This local inflammation may clear up quickly by the ciliæ recovering and moving on the invader; or, it may spread slowly or quickly to contingent areas, as so frequently happens. Again, certain gases and vapours, such as carbon-monoxide, sulphur-dioxide, pyridine, and numerous others, affect the ciliæ very powerfully, even in small percentages; so that, in conditions where these are present, hot stuffy rooms are an example, the ciliæ may move only very feebly, and in such conditions the bacteriæ may get an opportunity. This is especially likely to occur when a cold draught strikes one after inhaling such gases. This, on the top of the feebly moving ciliæ, will frequently allow the bacteriæ time to pour out their toxins before the ciliæ get moving again.

Besides these conditions, which apply to normal individuals, in nervous subjects interference and disturbance of movements of ciliæ is produced by fear, suggestion, and certain foreign substances which have a paralytic or delirious action on the ciliæ, a condition which does not result in normal individuals. Under such a condition of disordered local rhythm of ciliæ, irritation will result, with sneezing and other discomforts. As will be seen later, hay fever in the naso-pharyngeal mucous membrane occupies the same position as asthma in the respiratory tract proper.

The ciliæ of the respiratory tract, *i.e.*, from vocal cords downwards, are of immense importance in respiration. If their regular rhythm is disturbed, the effects are soon transmitted to the respirations. These become hurried; expiration or inspiration may become prolonged, and occasionally the disturbance may be so great as to cause death, as happens very occasionally in laryngismus stridulus and also in the condition known as acute suffocative catarrh. In this condition, large areas of ciliæ, as the result of toxæmia, are put out of action; brown froth and prune juice are brought up; signs of suffocation appear with sudden or gradual disappearance of cough reflex, due to total paralysis of ciliary movements.

In slight cases of disturbance, when only a very local effect is produced, the ciliæ are usually irritated by a foreign body, a plug of mucus, or bacteriæ. These affect movements of the ciliæ, with which they are in contact, causing a disturbance of the ciliary rhythm in the locality, and throwing them out of rhythm with the rest of the ciliæ. Irritation results, with production of mucus, and disturbance of respiration. There is a forcible expiration, which may or may not be preceded by a deep inspiration; in other words, cough results and, so long as the local ciliæ are prevented from moving to the normal rhythm, so long will cough result; even after the removal of the offending body, the ciliæ may take some time to recover and fall into the proper rhythm.

The production of mucus among the irritated ciliæ is an attempt to move along the irritant, as will be shown later. Nervous cough is due to disorder of ciliæ, and in such cases little or no extra mucus is produced.

Mucus is a glucosamine, and is slightly alkaline in reaction. It is precipitated by acid, so that any acid bacteriæ will solidify the mucus around it and, thus encapsuled, it is removed by cough and the movements of the ciliæ.

A second variety of mucus is faintly acid in reaction, and is precipitated by alkali, and thus alkaline bacteriæ are encapsuled and removed.

A third so-called mucus is really nucleo-albumen, and is produced after intense irritation, as occurs in tuberculosis.

The effect, then, of a local irritation is cough and production of mucus. Acute bronchitis is a further and more extensive condition. It often is an extension from the naso-pharynx, in which ciliæ have been paralysed by toxins, the toxins passing into contingent areas, and in these disorganizing the ciliæ. Sometimes it is due to direct inhalation of noxious gas, which acts, not in containing germs as was formerly thought in smells from bad drains, but in causing paresis and paralysis of the ciliæ. Noxious gases used in warfare have the same effect, and the prolonged inhalation of ether in some cases acts in the same way.

The result of such inhalation may result in paresis of ciliæ in a small area, or it may cause such extensive inflammation and œdema that the patient may die in 24 hours from suffocation, bringing up only froth. This is most likely to occur in gassing, and such a condition denotes paralysis of all ciliary movements.

In the case of a limited inflammation, as in acute bronchitis, extension is frequent. It may extend to fine bronchi, or it may attach these in first instance, causing capillary bronchitis. If the ciliæ of fine bronchi are completely paralysed in any area, mucus in vesicles is unable to be evacuated, and broncho-pneumonia is the result.

The general effects of the bronchitis will depend on the extent affected, and also on the degree in which the ciliæ are affected in that area. There may be simply cough and mucus; expiration may be prolonged; the respirations may be hurried. These effects are independent of rapid respiration due to asphyxia, which does not enter in these early cases; besides, these effects are to be observed in asthmatic and nervous cases. They are due to the effect of disordered action of ciliæ on respiration. The result of these changes in respiration is that an increased supply of oxygen is brought to the ciliæ, and this has a decidedly stimulating effect, usually restoring the lost movement at least in part. This is the condition in which oxygen is of use to the patient, either from the air or from a cylinder, and, if given to the ciliæ before they are quite paralysed, they may recover their movement. It is useless when the heart and pulse become affected, for asphyxia has then begun, and the ciliæ have, for the most part, been paralysed and destroyed, the accumulation of carbon-dioxide helping to produce this result.

Asthma is a neurosis, and although it may depend on bronchitis or tuberculosis when we have a combination of bronchitis and asthma, in its simplest form areas of ciliæ quickly pass into a disordered condition, either slowing or delirium. It may originate from nasal ciliæ or some other situation.

Such a disordered action spreads to a large area and causes interference with normal respirations, and "air hunger" results. The resulting asphyxia stimulates the respiratory centre, and the rapid breathing by the advent of increased oxygen restores the ciliæ, the movements of which may then return to normal or may again become disordered.

Laryngismus stridulus is a neurosis produced in a similar manner. Whooping-cough is due to the presence of bacteriæ and toxins, which irritate, paralyse, and destroy the ciliæ in several small areas. There are signs of pharyngitis, tracheitis, or bronchitis corresponding to the areas involved. In time, the bacteriæ and their toxins disappear, leaving areas of diseased and destroyed ciliæ. These areas interfere with normal movements of ciliæ, causing a neurotic disturbance of rhythm. Resulting from this, respirations are interfered with, a series of explosive coughs is followed by an inspiratory whoop. The frequency and severity of such spasms will depend, first, on the number and size of areas in which the ciliæ are destroyed, and the consequent amount of interference with the rhythm of the remainder of the respiratory ciliæ, and, secondly, on the nervous condition of the patient.

As the ciliæ recover or are replaced, the symptoms tend to disappear, but such areas for some considerable time may be hypersensitive, and on slight provocation they may throw the movements of the ciliæ out of action and the typical whoop may recur. It is curious to see how curiously correct the empirical treatment of the past appears to be. Alkalies stimulate the movements of the ciliæ and, of the alkalies, ammonia is by far the most powerful. They act, not by thinning the mucus but by stimulating the ciliæ, and these, by their movement and the irritation they then receive, cause increased output of mucus.

Oxygen also has a pronounced effect, and is more useful the earlier it is employed. Increase in respirations indicates toxins acting on ciliæ, which slow down.

This is of little consequence as long as there are no signs of lung involvement. As soon as these signs are present, oxygen, in addition to the other treatment, may be indicated.

Iodides, ipecacuanha, and antimony are useful, combined with alkalies, for they increase mucus and probably stimulate ciliæ. Camphor, eucalyptus, turpentine, and balsam groups also are exceedingly useful either internally or inhaled. If one may judge the value of a preparation by its universal use, camphorated oil causes the infant to inhale camphor fumes continually.

In considering the neurosis factor, belladonna and opium preparations are indicated in conditions in which there is little or no increase in amount of mucus. In conditions in which mucus *is* increased, these two drugs are strongly contra-indicated. The more mucus produced shows the desperate condition of the ciliæ, for it is only by increasing the amount that they can keep moving, and as long as they move they can get rid of the secretion. To dry up the secretion with belladonna is to deprive them of their only weapon against the invading bacteriæ. Opium acts in slowing down the movements of ciliæ directly, causing a sort of lassitude. This removes cough and lessens irritability and output of mucus. It has no direct effect on either heart or respiratory centre in ordinary doses. The action of this drug is just the opposite to what is required, and it should only be employed in conditions of pain, restlessness, and cough, such as pleurisy, in which mucus is not increased.

A Plea for Compulsory Restraint on the Use of "Domestic Measures" in the Administration of Medicine.

By H. O. GUNEWARDENE, M.B., B.S.
Late House Physician and House Surgeon, East London Hospital for Children, Shadwell.

MY suspicion of the inconstancy and indefiniteness of domestic measures, in the administration of medicine, was raised by my experience of errors arising from their use. This led me to a series of observations, the results of which, recorded below, show that there is little, if any, justification for their extensive use by either chemist or practitioner. On the contrary, they point to the striking fact, that an error of even 100 per cent. increase or decrease of a dose is always possible; they show that there is much error in the common supposition that a tablespoonful is equivalent to half an ounce, and a teaspoonful is equivalent to a drachm.

My observations have been made on spoons supplied to me by different people, and my findings are that spoons fall mainly into three different groups:

(*a*) Teaspoons.

1. Size A, half a drachm to one drachm when just full; 2. Size B, one drachm when just full; 3. Size C, equivalent to twice size B, or equal

to two drachms, when just full; with some occasionally varying in size and capacity between A, B, and C.

(*b*) Tablespoons.

1. Size A1 just full, equivalent to half an ounce; 2. Size B1, equivalent to twice A1 just full, equivalent to one ounce.

(*c*) Dessertspoons.

1. Size A2 just full, equivalent to two drachms; 2. Size B2, equivalent to twice A2 just full, equivalent to half an ounce. By just full, I mean only so full that the spoon can be held by an average person without any overflowing.

Of tablespoons, those I have commonly been given hold one ounce each, and of dessertspoons half an ounce each.

It may be asked, why then have noticeable ill-effects not been common? The reply is that the use of spoons of capacity equivalent to the dose prescribed, of marked bottles, and the tolerance to bigger doses than are prescribed, as well as the taking of smaller quantities than we advise, have prevented them; but ill-effects do occur, which are not only very unpleasant but sometimes even dangerous or disastrous.

In the administration of drugs to children, for whom dosage is a question of great importance, results contrary to our rational expectations occur more often than we suspect. In my own experience, excessive doses of castor oil, sugar and cream in feeds, of nux vomica and digitalis, insufficient doses of cough mixtures and too big doses of mixtures containing opium have been administered; a few cases of excessive action after mist. alba and liquid paraffin have also been noted. In one striking case of mine, a relapse during an attack of acute rheumatism (?) which, brought cardiac complications with it (pericarditis), was due, in my opinion, to a diminution of the dose of salicylates by half, through changing a marked for an unmarked bottle. This case is a questionable one, but if the unreliable measures were responsible for the complications, as I strongly suspect they were, the damage left behind is permanent. Further, is it not more than likely that some of the inconstant effects of drugs, and even some cases of idiosyncracy, may be traceable to overdoses arising from the use of spoons as measures?

After a careful consideration of the results of my limited experience, I maintain that—

1. The "teaspoonful" and "tablespoonful" are very indefinite and variable measures which ought not to be used.

2. Both practitioner and chemist should be enforced to adhere to the measures given in the British Pharmacopœia.

3. As an alternative, it should be insisted on, particularly in the out-patient department of hospitals, that all bottles given away are marked in some convenient way by the dispensing chemist, to signify the amount equivalent to the dose administered, unless measuring spoons or glasses are given or sold with the medicine administered.

4. Where circumstances compel the use of these measures, the patient should be shown the size of the spoon to be used, or, if it is in a private house, the practitioner should ask to be shown the spoon that is going to be used.

Practical Notes.

Treatment of Pneumonia.

1. *Specific Treatment.*—Formerly, this consisted of bleeding, emetics, and blistering. At the present time, serum-treatment is only suited to certain cases, so the treatment resolves itself into injections of camphor in oil, cold water, and adrenaline.

2. *Symptomatic Treatment.*—This concerns the patient and not the disease. General hygienic methods must be adopted with regard to the patient and his surroundings. The food should comprise milk, broths of various kinds, sugar, and *tisanes* sweetened with lactose.

The treatment of an ordinary case in an adult is directed, on *the first day,* to the side affected with the developing infection by means of counter-irritants, a cold-pack to the chest for four hours, injections of 8 to 10 cc. of camphor in oil, and stimulants.

On *the second day,* the disturbances in the alimentary system and dyspnœa must be dealt with by a purge with sulphate of soda, fractional doses of hydrochlorate of adrenaline by hypodermic injections, and wet cupping.

On *the third and fourth days,* the condition of the heart must still be carefully attended to, giving, if necessary, 5 to 10 drops of a 1 per cent. solution of digitaline in a draught containing from 4 to 6 g. of acetate of ammonia, or an injection of strychnine, 1 to 3 mg. each day.

If there is hyperpyrexia, the cold application to the chest must be replaced by baths, cooled by ice or at 82° F., or by a wet-pack every four hours.

On *the fifth and sixth days,* the treatment will be determined by the conditions present. The digitaline may be replaced by tincture of canella, or extract of cinchona in the acetate of ammonia draught, or an expectorant may be ordered containing acetate of ammonia and codeia. When the urine is scanty 60 drops of tincture of squills for the day should be added. If there is depression or anorexia, one or two injections each day of 250 cc. of glucose-serum (60 per cent.) should be given.

In *defervescence* and *convalescence,* the treatment will be adapted to the symptoms remaining. Full diet should be given at the earliest possible opportunity, beginning with semi-fluids.

3. *Treatment of Complications.*—Hyperpyrexia will call for cold baths every three hours, according to the condition of the heart and the strength of the patient. Large doses of camphor are necessary in the typhoid state, as well as intra-muscular injections of metallic ferments (collargol or lantol), inhalations or, better still, hypodermic injections of oxygen, and hypodermic injections of milk or of 5 per cent. peptone solution.—(*Journ. de Méd. et de Chir. prat.,* January 10, 1920.)

Reviews of Books.

The Art of Anæsthesia. By P. J. Flagg, M.D. Pp. 367. London: The J. B. Lippincott Co. 18s. net.

The second edition of this valuable work does not differ widely from the first, which appeared only three years ago. A chapter has been added upon the selection of anæsthetics, and there are some pronounced and perfectly justifiable expressions in the preface upon the practice of delegating the administration of anæsthetics to non-qualified individuals. Dr. Flagg is properly imbued with the responsibilites that rightly fall to the anæsthetist, and the whole work shows how he has prepared himself and is anxious to prepare others to meet them with the confidence that comes only from study and experience. Dr. Flagg makes an unusual, but a very practical, distinction of the stages of anæsthesia which he describes as induction, maintenance, and recovery; this is clinically preferable to the usual description of four stages which with modern methods of administration are generally quite impossible to differentiate. The book suffers a little from a tendency to repetition, but it is a most interesting work for the English reader, who will find described apparatus and methods with which he is unfamiliar and an absence of others with which he is already fully acquainted.

Handbook of Anæsthetics. By J. Stuart Ross, F.R.C.S. Pp. 214. Edinburgh: E. and S. Livingstone.

The author is to be congratulated upon having produced a book which, though very condensed, is eminently attractive. This is largely owing to the fact that he keeps in mind the relation of the facts of anæsthesia to those of medical knowledge generally, and is never in his writing so narrowly specialized as to be uninteresting or unintelligible to those who are not themselves specialists in anæsthetics. The physiological chapters are particularly good. The clinical evidences of the stages of anæsthesia are well described in detail; with the detail we find ourselves in disagreement, the eyelids are generally closed, not half open, in normal surgical anæsthesia. Mr. Ross does not allude to the cauthal tear, the value of which as an indication of light anæsthesia was so well demonstrated by the late Dr. Rutherford. Good descriptions are given of modern apparatus and the way to use it, and in a short chapter on the choice of anæsthetic the student is told what anæsthetic and what method of administering it is best suited to various special operations as well as to various types of subject. The book is well printed and illustrated, and we think that there is no small work on anæsthetics likely to be more acceptable to the student and practitioner.

Preparations, Inventions, Etc.

"OMNOPON-SCOPOLAMINE"—SCOPOLAMINE STABLE.

(London: The Hoffmann-La Roche Chemical Works, Ltd.,
7 and 8, Idol Lane, E.C. 3.)

Omnopon-scopolamine is a combination of omnopon, which contains the total alkaloids of opium in perfectly soluble form, with a pure lævo-rotatory salt of hyoscine, the hydrobromide of scopolamine, in sterile solution ready for hypodermic injection. Three strengths of the solution are now made available, the ampoules originally issued containing gr. $\frac{1}{3}$ of omnopon and gr. $\frac{1}{300}$ of scopolamine in each cc., and gr. $\frac{2}{3}$ of omnopon and gr. $\frac{1}{150}$ of scopolamine in each cc. The new introduction is a solution containing gr. $\frac{1}{3}$ of omnopon and gr. $\frac{1}{150}$ of scopolamine in each cc. Each ampoule contains 1·1 cc., so that exactly 1 cc. of the solution can be drawn up into the syringe and injected.

Scopolamine stable is a stable solution of the hydrobromide of scopolamine, put up in ampoules containing 1·1 cc. of the solution. Three strengths are supplied, containing respectively in each cc. $\frac{1}{150}$ gr., $\frac{1}{450}$ gr., and $\frac{1}{1000}$ gr. of scopolamine. Both these preparations are issued in boxes containing six and twelve ampoules

THE "PHYSIO" FARADIC WAVE APPARATUS.

(London: The Medical Supply Association, Ltd,
167–185, Gray's Inn Road, W.C.1.)

This apparatus is a modification of the Bergonié coil in such a way as to render that heavy instrument portable and at the same time suitable to provide every variety of current needed for the treatment of different conditions by the Faradic current. The painless current of the Bergonié coil is obtained with a fine muscular reaction so that both poles can be applied to the tongue, setting up violent contraction without the least pain. For conditions requiring a higher E.M.F., the apparatus has been fitted with a volt-selecting switch controlling four different windings, each of a different value. A sliding coil mounted on two parallel metal runners is provided for fine adjustments. A surging current can thus be produced, the maximum rise of which can be governed by a limiting stop so that each surge rises from the same minimum to the same maximum. The contact-breaker can be set to give a current as slow as 60 or as high as 3,000 impulses a minute. Two large dry cells are fitted, which are easily accessible. It is supplied in a mahogany case, the "on" and "off" switch being fitted in such a way that the apparatus cannot be closed whilst working, or without the case for fitting to switchboards and tables.

THE PRACTITIONER

APRIL
1920

Medico-Legal Notes

(*continued*).

By SIR JOHN COLLIE, C.M.G., M.D., J.P.

Medical Examiner to the London County Council ; late Director of Medical Services, Ministry of Pensions ; Home Office Medical Referee, Workmen's Compensation Acts, etc.

ANYONE who has spent weary hours in a stuffy County Court, may have noticed that the back benches are filled by a nondescript audience composed mostly of unemployed and unemployable. There is never a more attentive audience ; many are there to learn how to conduct themselves in the witness box when they are making a claim, and what line of conduct is best to pursue as regards resuming work during the long wait before their cases come into Court. Incidentally, they also learn how best to circumvent their *bête noir*, the medical man who is interested in malingering. The points which specially amuse and interest this motley mass are noteworthy. Their idea of humour and the evident sympathy shown towards men of their own class, even in palpably fraudulent cases, give a low comedy interest which helps temporarily to brighten an otherwise dreary experience.

Most working men believe that all pains, aches, and ill-health of any kind, with which they may be afflicted subsequent to an accident, must necessarily be due to it. The *post hoc ergo propter hoc* argument is simplicity itself; it is convenient, and saves no end of trouble. Lawyers appear readily to accept it; but, let us not forget, they are acting under instructions. A man meets with an accident; in the course of a thorough examination, probably many months later, a malignant growth is discovered. You will be asked: Is the condition the result of the accident? In the dead silence which follows the question, everyone is thinking that at last you have been put in a corner. I think the correct answer is: "Science has not yet discovered the cause of cancer; I cannot either deny or affirm that a malignant growth may be caused by an accident." But, while it is obviously fair to make this admission of the possible causal relationship, one should qualify it by adding: "But Science has not adduced conclusive evidence to support the theory that trauma is a factor in the ætiology of malignant disease."

County Court pathology is responsible for many grave injustices. In arbitration proceedings, the plaintiff should rightly have the benefit when there is a doubt, but, surely, it is wrong that he should get it when there is no doubt.

Many working men adopt a line of conduct which almost seems to necessitate their being branded as malingerers; but so to characterize them, even when the communication is absolutely privileged, should never be done without serious consideration, but only when prepared with incontestible evidence of fraud. However confident that the plaintiff is grossly exaggerating, is untruthful, and is attempting to deceive the Court, it is unwise to allege malingering unless it can be proved up to the hilt. Nothing, I find, pleases the plaintiff's counsel better than to get a medical witness to use the word "malingerer," for he knows that he can then appeal to the class prejudice of the jury. What, then, is a medical witness to say when invited to use the word "malingerer" in a case in which there is only a strong suspicion of malingering? One may say with truth that the plaintiff is not a malingerer. When asked: "If, as you say, this man is not a malingerer, and you disagree with his doctor's evidence, which is to the effect that he is still ill, why, then, does he claim that he is still not fit to return to work?" The reply is, that he is weak-willed, and will not face the necessary inconvenience, perhaps the small amount of pain, which he *must* have *whenever* he starts work. It should be explained that the plaintiff is irresolute, and has postponed, and will continue to put off, actually starting work because he feels that he is likely to suffer some inconvenience, which a reasonable man would put up with, and probably would last for only a few days, and that everyone who is accustomed to use his muscles, and has been off work for many weeks, has something to put up with at first.

Delay in returning to work, after the original disability has been recovered from, is a very frequent cause of an employer withholding payments, with the result that the case ends in Court. It is, therefore, a matter upon which a medical witness is frequently called upon to give evidence. Muscles which are accustomed to regular strenuous and daily exercise soon get out of condition. What one generally finds is that the workman's attention, and too often that of the medical attendant, is directed solely to the question of the healing of a wound or the union of a broken bone; the effect of immobilization upon muscle and other tissue is ignored. It is obviously unfair to expect an employer to pay indefinitely for a condition which is avoidable. No one is in "good training" who has been idle, whether the idleness is enforced or voluntary. It is well known that work is a better restorative for muscles than even the best massage; but how often is it really good? The great difficulty is that if a man does not really desire to become fit, no amount of massage or passive movements will ever bring him into a condition in which he can commence work without some inconvenience or some discomfort. This was well expressed by a County Court Judge, who said :—

> " I have often pointed out in previous cases that the sole object of the Act is to compensate workmen for the loss of wages so far as that loss is due to the accident. If a workman who has been injured has so far recovered that he is able to earn some wages, but makes no effort to earn those wages, his loss of those wages cannot be said to be due to the accident, but is simply due to the workman having made no effort to earn them. If, therefore, as in this case, the workman has chosen not to work, when the evidence shows that, so far as the accident is concerned, he is nearly as fit to work as he was before the accident, I cannot find that the loss of wages is entirely or even mainly due to the accident A workman can, according to the Act, be compensated for what he is unable to earn, but cannot be compensated for what he declines to earn " (*Lidderdale* v. *Robinson*, 132 L.T. 12).

It is clearly the duty of a workman, fit to undertake work of any description, to try and obtain it. An employer is not liable to pay full compensation, if the employee is able to work and earn *something.*

Under the Workmen's Compensation Act, the County Court Judge, acting as Arbitrator, has the duty put upon him of determining whether the plaintiff is or is not fit for work, and his decision upon this fact will not be interfered with by the Court of Appeal, if there is evidence to support it. Employers are only liable to pay compensation in respect of wages which a workman is *unable* to earn, and not on wages which he refuses to earn. The employer is not bound to provide him with work, nor to prove that he is able to obtain it. If he can prove that his late employee has so far recovered as to be able to undertake suitable work, it is the man's business to get it.

Employers are not liable for the results of unreasonable introspection after recovery. Cases are sometimes brought into Court in which an employee is in a state of nervous apprehension as the result of worrying or brooding over a recent accident, and entertains a fear of returning to that particular work because of the possible consequences; provided that such worry or fear would be unjustifiable in a reasonable person, the employer is not liable. The following case, quoted in my work on Malingering,* clearly sets out what was the judicial view in a case in which I was interested :—

> " In November, 1907, a woman relief-stamper met with an accident in the course of, and arising out of, her employment, whereby she lost the terminal phalanx of the middle finger and half the terminal phalanx of the ring finger of the right hand. She had been paid half wages for many months, when her employers applied to the Judge at the City of London Court to terminate the weekly payments on the ground that she was fit physically to resume her former work. They were willing, in the event of her returning to work, to pay her the full wages she had been receiving, or as much more as she could earn at her work. Two doctors gave evidence that the injured woman could do her work quite well. Judge Rentoul said that it was of the very greatest importance that it should be decided, once and for all, whether a workman or workwoman physically fit, but feeling nervous at working the same class of machine and therefore refusing to try it, should be considered unfit within the scope of the Act, and whether such a person could be compelled to go back to work or the compensation cease. He said he must hold that the compensation must cease, for the whole of the medical evidence showed the girl to be fit for her work in all respects, except in regard to timidity produced by the accident. It was possible that all her life she would shy at using the machine. Was she, then, to go on receiving compensation for ever ? The longer she kept off trying the worse it would be. A doctor, called for the workwoman, thought she might try to work, and the Judge directed that the payments be reduced to 1*d.* a week. He thought it was ' strong ' conduct on the girl's part that she had not tried to work." (*Pearson* v. *Pimms and Sons, Ltd.*, 126 L.T. 301).

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* *Malingering*, 2nd ed., Edward Arnold, London.

(*To be continued.*)

Medical Notes.

(SECOND SERIES.)

BY SIR THOMAS HORDER, M.D., F.R.C.P.
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DISEASES OF THE STOMACH.

DILATATION of the stomach is not in itself an indication for gastro-enterostomy. The indication for this procedure in a case of dilated stomach is pyloric obstruction, and in proportion to the degree of this defect which is present in any particular case will benefit follow the operation. If the case is one of primary " atonic " dilatation with ptosis, the result of short-circuiting the pylorus is nearly always disappointing, and not infrequently harmful.

(102) Pain is sometimes the sole symptom in a case of duodenal ulcer, and yet, if the pain has certain characters, a diagnosis may be made with considerable confidence. Much more hesitation is called for in the diagnosis of cholecystitis in similar circumstances. *In appendicitis, it is doubtful if a diagnosis based upon pain alone is ever justified.*

(103) Cardiospasm occurs under three different conditions: (i) in association with gastric " hyperacidity " and flatulence; (ii) as a neurosis; and (iii) in association with cancer of the cardiac end of the stomach. (i) is quite common; a point of importance in connection with it is that, if the pain is severe, which it not seldom is, and if the symptoms referable to the stomach are not very apparent, which they may not be, the source of the pain may be considered to be the aorta, and the pain may be thought to be anginal in character. (ii) occurs in women,

usually about middle age; it leads eventually to loss of flesh, even to emaciation, and to dilatation of the œsophagus; the patient often finds that she can swallow her food with relative comfort if she takes her meals alone. In (iii) the pain is usually much less severe than in (ii) and (i), and its occurrence is less erratic.

(104) Peptic ulcer is not cured by an operation performed to short-circuit it, and the surgeon who allows his patient to think so is either careless of the latter's welfare in the future, or learns nothing from experience. A short-circuit drains the stomach, allows physiological rest to the organ, facilitates healing of the ulcerated surface, and enables the patient to get much better results from appropriate changes in his diet and habits than he can do without these helps. *The operation should be regarded as being the first step in treatment and not the last.*

(105) Anorexia is an important, and often a very early, symptom in cancer of the stomach. It is also an important symptom in the differential diagnosis of this disease from simple ulcer, in which disease, though the appetite is often capricious (and sometimes excessive) it is rarely absent.

(106) A helpful point in the differential diagnosis of simple as against malignant pyloric obstruction is the condition of the blood; in simple obstruction, whatever the degree of emaciation—and this may be considerable—either anæmia does not occur or if present it is of slight degree, whereas in obstruction due to cancer anæmia is almost invariable and may be very marked.

(107) It is commonly taught that cancer of the stomach is usually preceded by such predisposing causes as gastritis, simple ulcer, etc., and that cancer

of the colon is preceded by constipation. The doctrine would seem to be based upon pure supposition; for though it is certain that, in a few cases, carcinoma supervenes upon chronic gastric ulcer, it happens much more frequently that the subjects of cancer of the stomach have been free from all forms of dyspepsia until the time the growth develops. The disease comes, as it were, " out of the blue." Similarly with cancer of the colon; more often than not the patient's first experience of troublesome constipation synchronizes with the development of the carcinoma. Moreover, both gastric dyspepsia and (especially) constipation are more common in females than in males, whereas the reverse is the case with cancer of the stomach and with cancer of the colon.

(108) Cancer of the stomach developing in a woman who is the subject of decided gastroptosis, may yield a tumour which is felt in the neighbourhood of the umbilicus, or even well below it. In these circumstances, despite the apparently bad prognosis indicated by the presence of a tumour, and by its size, an exploratory laparotomy should be undertaken, because this position of the growth is relatively favourable for complete removal. Such cases provide some of the few radical cures that have followed surgical treatment in the disease.

(109) *The only hope of radical cure in cancer of the stomach is afforded by very early diagnosis.* On no account should the observer wait for the development of a tumour. If a case of gastric dyspepsia, on all the evidence available, is considered to be probably of this nature, and if the patient continues to lose weight after 14 days of observation in bed with careful feeding, it is a sound proceeding to explore the stomach with a view to excision of the growth.

(*To be continued.*)

Recent Public Health Work.

By JOSEPH PRIESTLEY, B.A., M.D., D.P.H.
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I.—FORTY-SEVENTH ANNUAL REPORT OF THE LOCAL GOVERNMENT BOARD, 1917–18 (MEDICAL OFFICER).

THIS last Report may be described as the "swan song" of the Local Government Board, as well as the "swan song" of Sir Arthur Newsholme, the well-known medical officer of the Board, from which he has recently retired. The new Ministry of Health has now been established under the powers granted by the Ministry of Health Act, and Sir George Newman has stepped into the shoes of Sir Arthur Newsholme as the Chief Medical Officer of the newly-created Ministry, with the rank of an Under-Secretary—an important addition to the powers of the Medical Adviser to a public department.

In these circumstances, it was to be expected that some reference would be made to the work of the public health by the Local Government Board since the Board's establishment in 1871, and the future public health requirements for the country. This reference is to be found at the beginning of the Report under the two headings of (*a*) review of events since 1871, and (*b*) needs of the future. Under the former heading (review of events since 1871) attention is directed to the lack of knowledge of present methods of public health administration and of the progress which already has been made by local authorities in securing improved conditions of life for the people;

but a hope is expressed that, in the future, this lack of knowledge will no longer exist, especially now that women form an important element in the new electorate, and are already beginning to take an increasing interest in the administrative activities of local authorities in connection with maternity and child welfare. The improvements that have been effected in the public health since 1871 are not a reason for contentment, for there is still a large mass of avoidable or preventible morbidity and mortality existent. Success in the past must only be regarded as an encouragement and incitement to redoubled effort. Simplification of administrative machinery and extension of public health and medical work are alike needed.

Comparison of death-rates is the method adopted in the review, owing to other statistical information being either incomplete or wanting altogether. These death-rates for England and Wales are given as follows :—

Ages.	Death-rate per 1,000 Living at each Age-period in 1911–15.	Percentage Reduction in 1911–15 from Death-rate of 1871–80.
0	36·7	42
5	3·4	48
10	2·1	43
15	2·9	46
20	3·5	51
25	4·5	50
35	7·4	42
45	13·3	25
55	27·0	15
65	58·6	10
75	131·8	7
85 and upwards	262·9	15

Reduction in mortality goes, *pari passu*, with a reduction in morbidity (sickness and disablement). The expectation of life (or mean after-lifetime) at birth has, during the same period, been *increased* from 41·4 and 44·6 to 51·5 and 55·4, males and females respectively. Improved conditions of life have gone far to counterbalance the evils of urban aggregation, and these improved conditions of life have been brought about by pure water supplies, improvement in food supply, more adequate scavenging, drainage and sewerage, the clearing out of courts and alleys, better housing, street paving and sanitation generally, together with improved industrial hygiene (especially in the prevention of industrial dust and the reduction of hours of labour), the education of the people, police preventive work and that of voluntary agencies in regard to drunkenness and cruelty to children, etc. The financial aspect of these improvements is interesting and worthy of record, viz., total local taxation expenditure in England and Wales during the year 1871–72 was £31,185,471, as compared with £134,073,327 during the year 1911–12; whilst outstanding loans of all local authorities amounted to £69,461,087 in the year 1871–72, as compared with £556,571,139 in the year 1911–12. These financial figures are for *all* purposes, and do not include receipts for services rendered as deductions.

More than a quarter of a million fewer persons die annually than would die if the experience of the decennium 1871–80 continued, with a consequent enormous annual saving of sickness and disability, of loss of wages, of the production of dependent widows and orphans, etc. One of the chief causes of poverty is sickness. Under the latter heading of the reference in the Report to the work of the public health by the Local Government Board, since the Board's establishment in 1871—the heading dealing with the needs of the future—attention is drawn by Sir Arthur to the following points: (*a*) research; (*b*) extension of communal action; and (*c*) simplification and strengthening of administrative machinery. These ideas are fully worked out on pp. x to xx, especially the last-named (the proposed simplification and strengthening of administrative machinery). A Ministry of Health can only effect good in so far as it is able to improve the machinery of local and central health government, to amalgamate overlapping or incomplete agencies locally and centrally, and thus to simplify the stages through which proposals for reform need to pass. Greater driving power in the machinery is also necessary; temptations to inertia must be removed, and monetary inducements to better work rendered available. To these ends public opinion must be educated. Medical services should be readily available for all needing them, and not confined, as at present, to insured persons, to persons under the poor law, and to mothers and children under local schemes and to children of school age under educational authorities. In other words, every person born should have the right to command the best medical services throughout his or her life, including expert and consultative facilities, institutional treatment, and, of course, nursing advantages. Voluntary organizations and help are to be encouraged, to work in amalgamation and conjunction with official efforts. It must always be remembered as an axiom that public services concerned with preventive and clinical medicine cannot be separately administered without grave loss of efficiency. Prevention and treatment must go hand in hand, and the medical practitioner of the future must be a hygienist as well as a physician.

The new Ministry of Health has great potentialities. We now go back to the Report as a report dealing with the year 1917–18.

(a) *Maternity and Child Welfare.*

Maternity and child welfare work is progressing, and further statistics are set out showing the seriousness of still-births and infantile mortality amongst illegitimates. Artificial feeding of infants is dealt with, and the importance again emphasized of natural feeding, with the following interesting additions:—

1. Artificial feeding must be the last resort, and only used after trial of first artificially feeding the infant through its mother, *i.e.*, by feeding the mother with milk, thereby making her the physiological modifier and producer of milk for her infant.
2. *Partial* artificial feeding is better than *entire* or *whole* artificial feeding.
3. Four-hourly meals suffice, as a rule, from birth onwards, or at least from the end of the first month, the infant not being fed at all from 10 p.m. to 6 a.m.
4. Official pamphlets dealing with natural and artificial feeding (in the same pamphlet) should not be distributed, as a routine, by health visitors, visiting houses wherein births have been notified, but rather pamphlets dealing with the great importance of *natural* feeding only, and the necessity for securing the natural feeding by the feeding of the mother.

(b) *Prevention of Acute Infectious Diseases.*

Attention is drawn to the decline that continues in typhoid fever notifications, corresponding with a concurrent decline in typhoid fever mortality, even allowing for the fact that many adults (the age-period of the disease) are away on active service. Dysentery, too, has not caught on in Great Britain after the war (or during the war) as it has in Germany, Russia, and other countries. The causes are not far to seek, viz., small disturbance in Great Britain in the nature, amount, and quality of available food supplied to the civil population, Great Britain's insular position, precautions taken by the British Army to prevent the introduction of the disease, and the (speaking generally) good sanitary conditions that obtain in this country. Bacillary dysentery (the Flexner and Shiga bacilli) is the form of epidemic dysentery which needs constantly to be guarded against, whilst war conditions obtain. The tendency of all recent work is to minimize the relative importance of the factor of infection by *entamœba histolytica* in the production of dysentery epidemics. " Carriers " of this amœbic dysentery need not cause alarm. " Carriers " of bacillary dysentery must be strictly dealt with.

Louse-borne diseases—typhus and trench fever—are mentioned, with the significant addendum that no single case of either disease has been introduced, either from France or from more distant war areas, and yet no one disputes that lousiness and scabies have been introduced again and again. Malaria has appeared in this country indigenously, but the number of cases compared with the large number of malarious army and navy men who have returned home is, at present at least, very small, and points to the efficacy of the preventive measures which are being taken, including the compulsory notification of the disease.

Smallpox, epidemic jaundice, plague, cerebro-spinal fever, poliomyelitis, encephalitis, whooping cough, and measles are also dealt with.

(c) *Prevention of Chronic Infectious Diseases.*

Tuberculosis and venereal diseases are included under this section.

The remaining sections of the Report deal with the work of port sanitary authorities during the war, and the general work of the Medical Department.

To the Report are attached the following appendices :

1. Report on Enteric Fever in Bedlingtonshire Urban District, Northumberland.
2. Report on Two Outbreaks of Dysentery (Acute) in London

(Chelsea and Islington).

3. Statement on the Prevalence of Cerebro-spinal Fever in the Civil Population of England and Wales during 1915–16.
4. Copy of amended official Memorandum on Cerebro-spinal Fever.
5. Summary of Information as to Cases of Poliomyelitis occurring in 1916–17, with Observations on this Disease.
6. Report on the Work of Inspectors of Foods, 1917–18.
7. Report on the Operations of the Government Lymph Establishment, 1917–18.
8. Report on the Work of the Board's Pathological Laboratory, 1917–18.
9. Vaccinal Condition of Cases of Smallpox reported during 1917.
10. Application of Rationing System to Sanatoria.
11. Notification Statitstics of Ophthalmia neonatorum.
12. Notification Statistics of Still-births.
13. Port Sanitary Administration and Survey.
14. Circulars, Memoranda, and Orders issued 1917–18, relating to Questions affecting the Public Health.

All these appendices are interesting reading, and form the bases of the main Report and of the recommendations contained therein. Attention may be drawn to Appendix No. 2 on Dysentery in London, as a sort of model of what an official investigation should be. All the facts are tabulated after collection, and the conclusions drawn are obvious, and such as would be drawn by anyone who reads the Report carefully. The bacteriological data as set out are specially noteworthy. The Appendices Nos. 3, 4, and 5 bring official knowledge of cerebro-spinal fever and poliomyelitis up to date, whilst Appendix No. 13, on Port Sanitary Administration and the Prevention of Infectious Disease brought by shipping is a classic on this interesting subject.

II.—MATERNITY AND CHILD-WELFARE.

Looking back five years it seems almost incredible that the point of view taken to-day in connection with the subject of maternity and child-welfare should be possible. The subject is now practically in the hands of preventive medicine. Ante-natal clinics, natal clinics, and post-natal clinics are now departments of Public Health Administration for which Medical Officers of Health are primarily responsible to Sanitary Authorities, whose duty is the health of the people. Municipal maternity and child-welfare schemes are springing up and flourishing in all directions under what has been aptly described by an ex-President of the Local Government Board as the fertilizing influence of Government grants. Expectant mothers are dealt with first, and, doubtless in due course, the next forward move will be for the Government to deal with expectant brides and bridegrooms; it is merely a matter of time from the present point of view of the duties of the State. Preventive methods in pregnancy and maternity have come to stay, and are the direct outcome of the Notification of Births (Extension) Act, 1915, and the Maternity and Child Welfare Act, 1918, together with the various Midwives Acts.

Pro rata with the above administrative changes and advances, the status of the official health visitor is to be improved. Draft regulations have been issued by the Board of Education to local authorities, dealing with the training of health visitors. The certificate of the Central Midwives Board becomes essential for health visitors connected with midwifery. In all cases, candidates must possess a good general education, and, in addition, must be certificated after passing special examinations, to be held after attendances upon the courses of special training. The course of training is one of two years for candidates without previous training, and of one year in the case of fully trained nurses, of women with experience of health visiting, or of those possessing a University degree or its academic equivalent. All *salaried* Health Visitors appointed after a certain date (of which due notice will be given) must have obtained the certificate described in the Regulations of the Board of Education, and grants will then be available in respect of salary in such cases, but will not, as a rule, be available in respect of salaries of newly-appointed Health Visitors who have only the qualifications of a midwife or a Sanitary Inspector, unless such Health Visitors have previously rendered satisfactory service as such in other districts.

Unpaid voluntary workers are acknowledged under the Regulations as rendering valuable assistance to the work of Maternity and Child-welfare Centres. Such voluntary workers must be experienced and competent, and some training is desirable for them also; but they need not necessarily possess certificates given after formal courses of training.

Further, until such time as the Ministry of Health considers that there have become available sufficient numbers of candidates, who have been trained and certificated as required under the Regulations, women who are now acting as Health Visitors, and women who may be appointed during the intervening period as Health Visitors, will not be required to have taken either the full (two years) or the shortened (one year) course of training as a condition of repayment of half their salaries from the maternity and child-welfare grants given by the Ministry of Health, which, however, expresses a hope that local authorities will afford facilities to such Health Visitors to take an approved course of training, where they desire to do so, and where the respective Medical Officers of Health are of opinion that the course of training would increase the Health Visitors' efficiency.

Grants are also available from the Board of Education in aid of the training of women to become Health Visitors.

Ante-natal clinics are as important as natal and post-natal clinics, and closely associated with them all should be dental clinics, day nurseries, and even night nurseries. Maternity homes, where women of the poorer classes can be confined for a nominal fee, and away from their own crowded and sanitarily unsatisfactory houses, are also a prime necessity in all working-class and poor neighbourhoods. For all these purposes, not only are properly trained health visitors, nurses, and midwives needed, but also properly qualified and experienced medical men and women. In addition, a new kind of official is being introduced, called the "home-help," whose duty it is to take charge of the *home* of the woman during her confinement and after (for a short period), whether such confinement take place at the house or away from the house at a maternity home or institution. In this way, the children are looked after and sent off properly to school (if of school age), meals provided for the husband and the other inmates, and the premises generally kept clean and tidy, thereby relieving the mother from much worry and anxiety. Where these new officials have been tried, they have justified the experiment. "Home-helps" are a necessity, as will be admitted by all who have had experience of the usual chaotic conditions that may obtain in an ordinary working-class or poor-class dwelling or flat, when a confinement takes place and during the necessary convalescence of the mother.

The new Maternity and Child-Welfare Act, 1918 (passed August 8, 1918), must tend to boom maternity and child-welfare. Under this Act maternity and child-welfare committees can be appointed—each committee to include at least two women members.

III.—VENEREAL DISEASE AND ITS PROPHYLAXIS.

A memorandum on prophylaxis against venereal disease has been issued by the Inter-departmental Committee on Infectious Disease, during Demobilization. It is only one of many that will, probably, be issued in the future, and its publication, at the present time, is on account of the urgency of the subject-matter. Should certain methods of prophylaxis against venereal disease (known as the "packet" system) be made available to the civil community, in view of their reported success amongst combatants during the war? This is the only question which the memorandum answers, and the answer is in the *negative.* As a corollary, it follows that the members of the Inter-departmental Committee are not satisfied from the evidence submitted to them that the *reported* success amongst combatants during the war of such methods of prophylaxis has been proved. In other words, the *report* of such success is not justified by the facts. The Government is, therefore, not advised to encourage officially the sale of "packets," containing means of prophylaxis for use by the individual, before or after exposure to infection—the view of the Committee being set out in the following words:—

> "However careful the instruction, and however scientifically effective the prophylaxis in itself might be, if used under proper conditions, it frequently fails to protect when applied by the individual, even when this self-application is supported later by additional skilled treatment. The consumption of alcoholic liquors, carelessness, natural excitement, forgetfulness, or ignorance, have been the cause of numerous failures, even amongst those troops where the use of such prophylactic measures has been most efficiently organized and taught."

IV.—INFLUENZA AND ITS PREVENTION.

No review of recent public health work would be complete without a reference to the serious epidemic, or rather pandemic, of influenza that has, during the last 12 months, swept over the world. The influenza started in China or Russia, where it is *endemic*, became *epidemic*, spreading to neighbouring countries along the ordinary channels of human intercourse, and from persons to persons, and finally took on *pandemic* proportions, passing over the whole world, few, if any, places escaping. The seriousness of the disease is shown by the mortality returns, which were enormously increased everywhere. As an example the Borough of Lambeth may be taken: a fourth of the total deaths from all causes were due to influenza, directly or indirectly, and the age-period that suffered most was 25 to 40 years, though the age-periods 45 to 60 years and over 60 years also suffered severely. The sexes suffered equally, and the disease was spread generally throughout the borough, affecting no particular area or areas, the inner, more crowded districts suffering equally with the outer, less crowded districts. The disease appeared in three forms: (*a*) nervous,; (*b*) catarrhal (bronchitis and pneumonia), and (*c*) gastric; but the catarrhal form was the commonest. Pfeiffer's bacillus, the streptococcus, and the pneumococcus were isolated, and it is probable that these germs (separately or together) were the cause of the disease. Secondary cases were common, showing that the disease spread rapidly from person to person, the infection being most virulent during the first few hours (lasting in a less virulent form up to two days) of the disease. Whole households were *hors de combat* from the disease at one and the same time, and the staffs of workshops and factories practically depleted to half-shifts in the course of a few days. Second attacks of the disease in the same persons were very few, if any. The incubation period is 48 hours. Morbidity must have been enormous, judging from mortality.

As to preventive measures, the Royal College of Physicians of London deemed the epidemic of such importance as to warrant the issuing of an authoritative statement on the subject, and this statement was adopted by the Local Government Board officially and circulated by the Board amongst all Medical Officers of Health, in addition to the Board's other circulars dealing with the epidemic.

The total *real* damage that the influenza has done will be shown when the whole of the statistics for the different countries have been issued, giving the total deaths and the percentage deaths, the latter being the more important because they will indicate to what extent the epidemic has increased the total general death-rate; an increase that may or may not prove to be as great as the layman and even some experts anticipate. Many other facts will be tabulated.

Public authorities have difficulties of administration in dealing with an influenza epidemic, on account of the very short incubation period of the disease and the rapidity with which the disease spreads from person to person. The ordinary means of prevention do not apply in the sense that they apply to a disease like smallpox or scarlet fever, viz., (*a*) prompt notification; (*b*) isolation in a hospital; (*c*) disinfection; and (*d*) medical inspection of contacts. It must rather be left to the individual to apply such measures to himself and his immediate surroundings, leaving to the Public Authorities the educational side and the preventing of overcrowding in public places, vehicles, schools, etc., as well as in private premises used as common lodging houses and dormitories, etc., and the arranging for the proper ventilation at stated intervals of all such public or *quasi* public places. The Local Government Board introduced orders with a view to such ventilation being effected, known as the Public Health (Influenza) Regulations, 1918, and the Public Health (Influenza) Regulations (No. 2), 1918, under which it is provided that, where the public are admitted to a place of public entertainment (as defined in the Regulations), the entertainment shall not be carried on for more than three hours consecutively, except in the case of a cinematograph exhibition, when the period of three hours may be extended to four hours;

and further, that there shall be an interval of not less than 30 minutes between any two entertainments given at a place of public entertainment (as defined in the Regulations), to which the public are admitted, such interval to be for the purpose of the premises being effectually and thoroughly ventilated. In the case of cinematograph exhibitions, children from a district wherein a public elementary school has been temporarily closed on account of the prevalence of influenza, shall not be admitted to such exhibitions in that particular district during the continuance of the closure of any such school. The Regulations came into force on November 25, 1918, to continue in force until revoked by Order of the Board, and apply to England and Wales.

School closure or exclusion, exclusion from or closure of places of public entertainment, exclusion from occupations, provision of proper nursing and medical assistance and of the more recently introduced domestic assistance or home-help, are also matters that fall amongst the duties of public authorities in connection with the carrying out of preventive measures to be taken in dealing with influenza outbreaks, which may be so widespread as to be epidemics or even pandemics. It must not be forgotten, however, how important are the personal or individual's preventive measures.

Therapeutic Measures in Influenza.

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ATTEMPTS to discover a specific remedy for a disease, before its causative organism is known, must of necessity introduce the element of chance. This, however, does not mean that the *modus ab experimento* should be condemned, as is shown by the brilliant results which have attended its use in the treatment of malaria with quinine, which was in vogue in the middle of the seventeenth century, whereas the specific parasite was not recognized until towards the end of the nineteenth century. Thus, if it may be argued that the cause of influenza has not yet been discovered, still more may it be maintained that the cure is not yet known.

The more one reads of satisfactory modes of treatment, the greater the disappointment when one watches a case, that has had a highly vaunted treatment begun early and conscientiously carried out, proceed to a fatal issue, whereas another, that has had only symptomatic treatment, recovers.

SYMPTOMATIC TREATMENT.

The patient should be in bed, from the first day of the disease; the earlier, the better is his hope of recovery. Further than this, symptomatic treatment is similar to that adopted in any acute infectious disease.

SPECIFIC TREATMENTS.

An effort was made to test the value of certain specific methods of treatment. These were (*a*) potassium iodide and creosote, (*b*) vaccines, (*c*) salicin, (*d*) perchloride of mercury, (*e*) blood transfusion.

(a) *Potassium Iodide and Creosote.*—These drugs were administered in the following mixture:—

℞	Creosoti	♏ v.
	Potass. Iodid.	grs. x.
	Spirit. Vini. Rect.	♏ xv.
	Extract. Glycyrrhiz. Liq.	♏ xx.
	Aquam Chlorof.	ad ℥i

℥i quartis horis sumend.

Of the first 30 cases treated by this method, four died—all with broncho-pneumonia. The method cannot, therefore, be considered successful.

(b) *Vaccines.*—An investigation was then made during December 1918 to determine the effect, if any, of the administration of small doses of the Millbank prophylactic influenza vaccine after the onset of the disease. The results were judged by (*a*) the development of a moderate or severe bronchitis, (*b*) the development of broncho-pneumonia, (*c*) the development of toxæmia or the occurrence of death.

Bronchitis, in a mild form, affords no criterion of the severity of the infection, for practically all cases, on careful physical examination, show alterations in signs in the lower lobes of the lungs, indicating an exudative process into the alveoli or finer bronchioles.

The Vaccine was a mixed one, containing (B. Influenzæ, 60 millions; Streptococci, 80 millions; Pneumococci, 200 millions) in each cc.

Taking one cc. as the prophylactic dose (hereafter referred to as P.D.), the doses adopted for treatment were as follows:—

First Injection.—1/50 P.D. (B. Influenzæ, 1·2 millions; Streptococci, 1·6 millions; Pneumococci, 4 millions).

Second Injection.—The next day but one, provided that no serious reaction occurred, a larger dose, 1/30 P.D., was given (B. Influenzæ, 2 millions; Streptococci, 2·6 millions; Pneumococci, 6·6 millions).

Third Injection.—Again, after a day's interval, if the temperature was still raised, an increase was made, and 1/20 P.D. given (B. Influenzæ, 3 millions; Streptococci, 4 millions; Pneumococci, 10 millions). These injections were all given subcutaneously.

As very slight reactions, either local or general, appeared to ensue from these small doses, the later cases in the series were given larger doses, as follows:—

First Injection.—1/4 P.D. (B. Influenzæ, 15 millions; Streptococci, 20 millions; Pneumococci, 50 millions).

Second Injection.—1/3 P.D. (B. Influenzæ, 20 millions; Streptococci, 26·6 millions; Pneumococci, 66·6 millions).

Third Injection.—2/3 P.D. (B. Influenzæ, 40 millions; Streptococci, 53·2 millions; Pneumococci, 133·2 millions.

27 cases were vaccinated, and 27 observed as controls.

The cases were taken alternately as admitted—one being vaccinated and the next serving as a control; the results are shown in the tables.

Vaccinated.

Case No.	Day of Disease of 1st Vaccine.	Toxic Type.	Bronchitis.	Broncho-pneumonia.	Doses of Vaccine.	Result.
1	2	—	Slight	—	1/50 and 1/30 P.D.	Convalescent.
2	3	—	,,	—	1/50 and 1/30 P.D.	,,
3	4	—	Moderate	—	1/50, 1/30, 1/20 P.D.	,,
4	3	Yes	,,	—	1/50, 1/30, 1/20 P.D.	,,
5	4	—	Severe	—	1/50, 1/30, 1/20.	,,
6	5	—	Moderate	—	1/50, 1/30	,,
7	5	—	,,	—	1/50, 1/30, 1/20.	,,
8	4	—	Slight	—	1/50, 1/30, 1/20.	,,
15	6	No	,,	No	1/50, 1/30 P.D.	,,

Vaccinated—cont.

Case No.	Day of Disease of 1st Vaccine.	Toxic Type.	Bronchitis.	Broncho-pneumonia.	Doses of Vaccine.	Result.
19	4	No	Severe	Yes	1/50, 1/30, 1/20 P.D.	Convalescent.
21	3	No	Slight	No	1/50, 1/30, 1/20 P.D.	„
26	4	No	Moderate	No	1/50, 1/30, 1/20 P.D.	„
27	4	No	Slight	No	1/50, 1/30 P.D.	„
30	4	No	„	No	1/50, 1/30, 1/20 P.D.	„
31	3	Yes	Extreme	Yes (Empyema).	1/50, 1/30, 1/20 P.D.	Death.
32	2	No	Slight	No	1/50, 1/30, P.D.	Convalescent.
33	2	No	Moderate	No	1/50, 1/30, 1/20 P.D.	„
37	3	No	Severe	No	1/50, 1/30, 1/20 P.D.	„
40	3	No	Slight	No	1/50, 1/30, 1/20 P.D.	„
44	2	No	„	No	1/50, 1/30 P.D.	„
46	2	No	„	No	1/4, 1/3, 2/3 P.D.	„
48	1	No	„	No	1/4, 1/3 P.D.	„
51	2	No	Moderate	No	1/4, 1/3, 2/3 P.D.	„
53	3	No	Severe	No	1/4, 1/3 P.D.	„
55	1	No	Slight	No	1/4, 1/3 P.D.	„
20	4	No	„	No	1/50, 1/30, 1/20 P.D.	„
22	4	No	Severe	No	1/50, 1/30, 1/20 P.D.	„

Unvaccinated Controls.

Case No.	Toxic Type.	Bronchitis.	Broncho-pneumonia.	Result.
10	No	Slight	No	Convalescent.
11	No	„	No	„
12	No	„	No	„
13	No	„	No	„
14	No	„	No	„
16	No	„	No	„
17	No	„	No	„
18	No	„	No	„
23	Yes	Severe	Yes (Empyema).	„
24	Yes	„	Yes	„
25	No	Moderate	Yes	„
28	No	Slight	No	„
28A	No	Moderate	No	„
29	No	Slight	No	„
34	No	Severe	No	„
35	No	„	No	„
36	Yes	„	Yes	Death.

Unvaccinated Controls—cont.

Case No.	Toxic Type.	Bronchitis.	Broncho-pneumonia.	Result.
38	No	Moderate	No	Convalescent.
39	Yes	Severe	No	Death.
42	No	Moderate	No	Convalescent.
43	No	Slight	No	,,
45	No	,,	No	,,
47	No	,,	No	,,
49	No	,,	No	,,
50	No	,,	No	,,
52	No	,,	No	,,
54	No	Moderate	No	,,

The two series of cases may be compared as follows:—

	Moderate or Severe Bronchitis.	Extreme Toxæmia.	Empyema.	Broncho-pneumonia.	Convalescent.	Died.
	per cent.	per cent.	per cent.	per cent.	per cent.	per cent.
Vaccinated -	48	7	3·7	7	96·3	3·7
Unvaccinated	41	14	3·7	11	92·6	7·4

Conclusions as to Vaccine Treatment.—The figures tend to show that vaccinated cases do better than unvaccinated, but the series is too small to warrant any definite statement; certainly, the vaccine did not produce any strikingly beneficial results.

Wynn[1] writes enthusiastically in favour of larger doses of the mixed vaccine as a curative agent. He starts with 80 to 100 million of each of "influenza" bacilli, streptococci, and staphylococci.

He records three cases inoculated within the first 24 hours of the disease, in which the temperature fell, as by crisis, to normal shortly afterwards, and refers to other uncomplicated cases which showed a similar fall in temperature on the second, third, and fourth days. He states: "I have seen no similar charts in uninoculated cases."

I have seen many such; perhaps Figures 1 and 2 will suffice to show how this may occur early, apart from broncho-pneumonia, without any specific treatment; or Figure 3 when the fall occurred during treatment with mercury.

Wynn's more severe cases are also unconvincing, for he does not give the mortality of those which received the first inoculation later than 48 hours

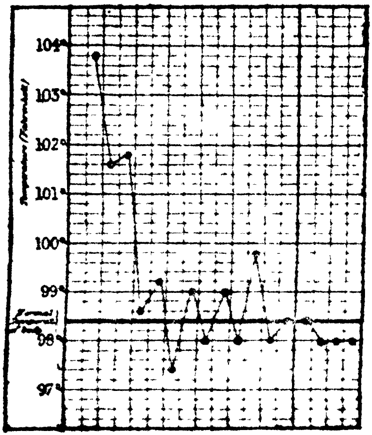

FIG. 1.

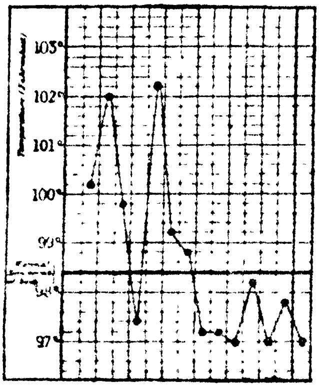

FIG. 2.

from the onset of the disease. The more cases one sees, the more difficult becomes the problem of *post hoc* and *propter hoc* in reference to sudden falls in temperature and the exhibition of different remedies.

Further, Wynn does not record any control cases. My own conclusions are that the vaccine method has not yet been shown to be in any way superior to the symptomatic method of treatment.

(c) *Salicin.*—Turner[2] recalls the fact that in the previous epidemic (1891) he treated 250 cases of influenza with large doses of salicin, and in the ensuing five or six years upwards of 2,000 by giving salicin gr. xx every hour "without any sequelæ, complications, or loss of life." He claims that these large doses of the drug cure the disease rapidly (average 36 hours), and so check its infectivity and spread.

Pollard[3] writes to support Turner, and states that he has given salicin, "although not quite so heroically," but he does not inform us what measure of success has attended his treatment. With a view of determining whether I could have equally successful results a series of 20 cases were treated with salicin, and 20 used as controls.

Doses of gr. x were given every hour for 36 hours, then every two hours for 24 hours, then every four hours for 24 hours, and then three times a day. The patient should, therefore, have come well under the influence of the drug; he was not wakened during the night, if asleep when the dose was due. The results appear in the tables below :—

Salicin Cases.

Case No.	Day of Disease on which Treatment commenced.	Duration (in Days) of Pyrexia.	Broncho-pneumonia.	Result.
96	2nd day	15 days	Yes	Convalescent.
97	2nd ,,	8 ,,	No	,,
99	4th ,,	10 ,,	No	,,
100	4th ,,	11 ,,	No	,,
101	4th ,,	6 ,,	Yes	,,
103	2nd ,,	6 ,,	No	,,
105	2nd ,,	6 ,,	No	,,
106	2nd ,,	7 ,,	No	,,
107	4th ,,	8 ,,	No	,,
108	3rd ,,	7 ,,	No	,,
117	2nd ,,	3 ,,	No	,,
121	4th ,,	5 ,,	No	,,
124	6th ,,	16 ,,	Yes	Death.
125	4th ,,	6 ,,	No	Convalescent.
126	7th ,,	9 ,,	Yes	Death
88	5th ,,	13 ,,	Yes	Convalescent.
89	8th ,,	19 ,,	Yes	Death.
90	8th ,,	14 ,,	Yes	,,
91	3rd ,,	20 ,,	Yes	Convalescent.
91A	3rd ,,	5 ,,	No	,,

Mortality, 20 per cent. ; average duration of pyrexia, 9·7 days ; broncho-pneumonia, 40 per cent.

Salicin Control Cases.

Case No.	Duration of Pyrexia.	Broncho-pneumonia.	Result.
98	7 days	Yes	Convalescent.
102	4 ,,	No	,,
109	9 ,,	No	,,
110	7 ,,	No	,,
111	5 ,,	No	,,
112	5 ,,	No	,,
114	6 ,,	No	,,
115	3 ,,	No	,,
116	8 ,,	No	,,
118	7 ,,	No	,,
119	16 ,,	No	,,
120	8 ,,	No	,,
122	6 ,,	No	,,

Salicin Control Cases—cont.

Case No.	Duration of Pyrexia.	Broncho-pneumonia.	Result.
123	5 days	No	Convalescent.
127	7 "	No	"
92	8 "	Yes	Death.
128	7 "	No	Convalescent.
68	10 "	Yes	"
71	17 "	Yes	"
77	5 "	Yes	"

Mortality, 5 per cent.; average duration of pyrexia, 7·4 days; broncho-pneumonia, 25 per cent.

Conclusions as to Salicin Treatment.—The mortality of cases treated with salicin was four times as great as those who received no specific treatment. The average duration of pyrexia was 2·3 days longer in the cases treated with salicin. Broncho-pneumonia occurred 1·6 times more often in the cases treated with salicin. But the figures cannot be treated quite as literally as this, for all the cases that died had broncho-pneumonia on admission, before the treatment could be begun. No. 96, however, developed broncho-pneumonia despite early saturation with salicin, but, after being desperately ill, recovered.

I think it may therefore be stated that salicin is of no avail once broncho-pneumonia has begun, the patient may have the drug in large doses and yet die eight or ten days later. Whether or not salicin, given in these big doses during the first 10 hours of the disease, would prevent pulmonary complications or death in every case has not been shown, because it has been impossible to see the cases sufficiently early in every instance. Turner[4] treated 335 cases since October 1918. " Every one ended in complete recovery with no complications, without a single death." These cases followed in unbroken series; upwards of 2,300 treated since 1889. He was sufficiently fortunate to be able to start the treatment in every case within 18 hours of the onset of the first symptom, which perhaps introduces the possibility of error in the diagnosis of some of them.

From the symptomatic standpoint salicin appears to relieve the muscular pains very much more efficiently than its *confrères* aspirin or sodium salicylate. Once broncho-pneumonia has developed, which may be very early, salicin cannot be considered as a certain cure; in fact, it is probably of no greater value than other remedies extolled from time to time.

(d) *Perchloride of Mercury.*—If, as there is evidence to show,[5] influenza is a septicæmic disease, it is logical to determine the effect of the intravenous administration of antiseptic substances.

Siciliano,[6] of Florence, recommended to Ferrarini intravenous injection of perchloride of mercury. Ferrarini[7] advocates the intravenous injection of 1 c.gm. of perchloride of mercury dissolved in 1 cc. normal saline daily for four to five days.

Having so far failed to find a satisfactory method of treatment, I determined to put this to the test. At first I was reluctant to give the drug by this route, fearing it might damage the renal or intestinal epithelial cells.

Since satisfying myself that no harm was done, I notice that Humbert,[8] on account of the possibility of this danger, has given intravenous injections of hexamine in preference to mercury; but it appears to me doubtful whether hexamine will exert its antiseptic action in a medium which, on account of its reaction, does not permit of the liberation of formaldehyde.

I have treated *a series of* 10 *cases* with daily intravenous injections of 1 cc. of normal saline in which 1 c.gm. of perchloride of mercury was dissolved.

The cases were all acutely ill, and were as follows :—

1. Case No. 130.—Treatment begun on the seventh day of disease. Temperature, 102° to 103° F. No broncho-pneumonia. Urine before treatment began contained albumen in moderate

quantity (0·36 per cent.); no red or white blood cells and no casts; a few epithelial cells, and gram-positive cocci and coccobacilli. One c.gm. perchloride of mercury dissolved in 1 cc. normal saline was given intravenously. The pulse and respirations were counted hourly for six hours after the injection, but were not affected. The temperature commenced to fall by lysis to normal, and after repeating the injection on three successive days the temperature was normal (tenth day of disease). On the thirteenth day of disease the temperature began to rise again, and one more injection was given; the temperature then fell to normal.

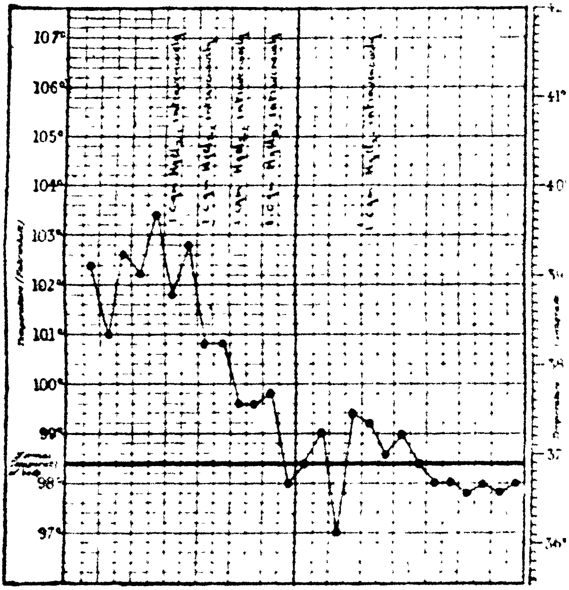

FIG. 4.

The urine was examined daily, the albumen steadily diminished, being 0·02 per cent. the day after the first injection, and too little to estimate the next day. No casts nor blood cells appeared in the urine, and the patient made a rapid and uninterrupted recovery. (*See* Fig. 4)

2 CASE NO. 132—Treatment begun on the sixth day of disease. Patient severely ill, cyanosed, and bronchitic. No broncho-pneumonia. Urine before treatment contained no albumen. One c.gm. perchloride of mercury given intravenously. Temperature rose to 105° F. that evening, but there was no rigor, and no acceleration of pulse or respirations during the six hours following the injection. On the next day the injection was repeated. The temperature fell to 103° that evening, and the urine contained no albumen. Injection repeated the next day, and the temperature fell as by a crisis to 98°, between the second and third injections. Two more injections were given on the succeeding days, and the temperature remained down. This patient had an attack of diarrhœa, with passage of bright blood

on the second and third days of the injections. (*See* Fig. 3.)

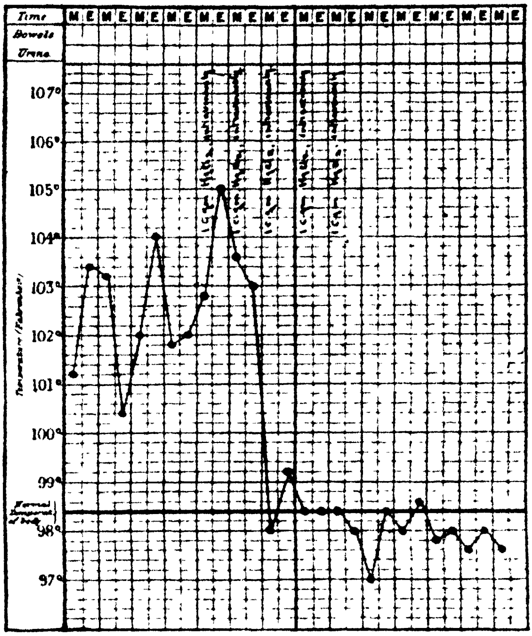

FIG. 3.

3. CASE NO. 124.—The treatment was begun as a last hope on the twelfth day of the disease when the patient was desperately ill with intense cyanosis, bilateral broncho-pneumonia, and general bronchitis. The first injection was given in the evening of the twelfth day, with temperature of 102°·8 F.—the temperature had fallen next morning to 100°·2, and the patient seemed somewhat better. The injection was repeated. The next two days the dose was increased, 1·5 c.gms. of perchloride of mercury being given on each occasion. The patient did not improve, and died on the morning of the sixteenth day. No albumen appeared in the urine, and although there had been an attack of enteritis four days before the first intravenous injection, it did not occur again during the treatment.

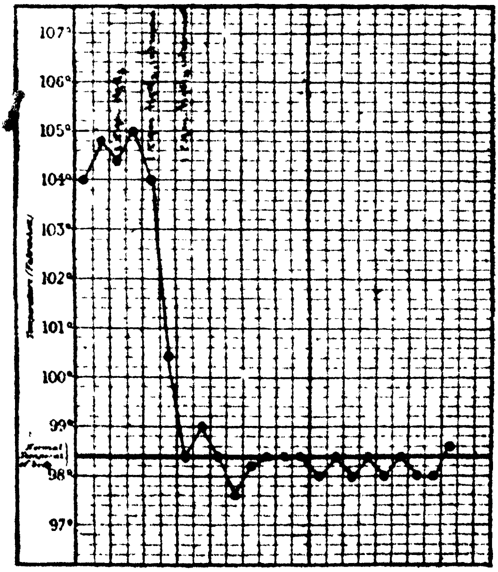

FIG. 5.

4. CASE NO. 159.—Treatment begun on the fourth day of illness, patient being severely ill with broncho-pneumonia and a continuous temperature of 104° to 105° F. After the second injection of 1 c.gm. perchloride of mercury, the temperature fell by crisis to normal and did not rise again. One more injection only was given. The urine before and

after treatment contained no albumen, and only a few epithelial cells. No enteritis occurred. (*See* Fig. 5.)

5. **Case** No. 162.—Treatment begun on the fifteenth day of illness, during a relapse in the course of disease and development of broncho-pneumonia. Temperature, 104°. Respiration, 50. After two injections the temperature fell to normal; three more injections were given at daily intervals, and the temperature settled down after once rising to 100°·8 F. The urine contained no albumen, before or after treatment. There was some diarrhœa before the treatment commenced, but it did not persist. (*See* Fig. 6.)

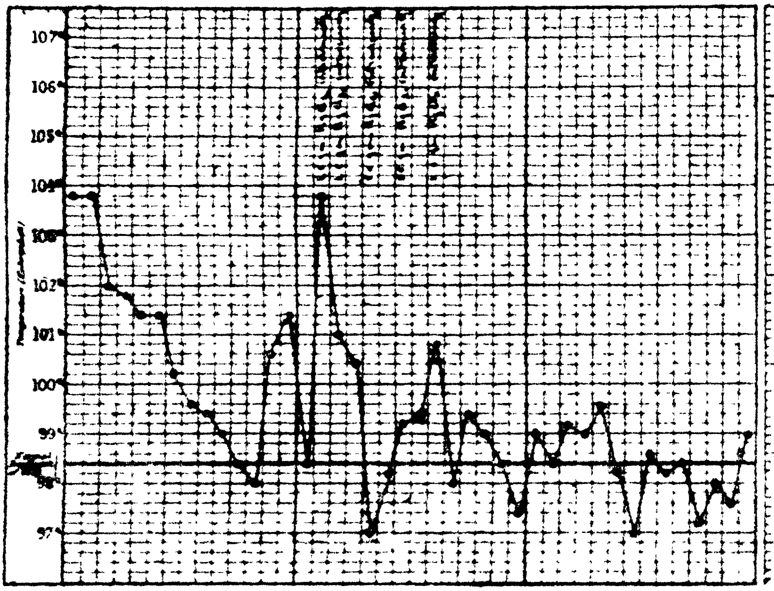

Fig. 6.

6. **Case** No. 164.—Treatment begun on the seventeenth day of illness, during a relapse. Patient was then very ill with pericarditis and bronchitis. Temperature, 101°·2 F. Five daily injections of 1 c.gm. perchloride of mercury were given without any appreciable results. The temperature finally came to normal on the fortieth day of illness—after a small sterile pleural effusion had developed.

7. **Case** No. 179.—Treatment begun on the fifth day of disease. Temperature 102°·6. Patient very cyanosed, bronchitis present but no apparent pulmonary consolidation. Daily intravenous injections of 1 c.gm. perchloride of mercury given. Temperature fell in three days from 104°·2 F. to 97°·6 F., and the patient rapidly improved. The urine contained no albumen before or after treatment. (*See* Fig. 7.)

8. **Case** No. 180.—Treatment begun on the ninth day of disease. Patient had a very toxic appearance, was cyanosed and drowsy.

Temperature, 103°·2; he had acute bronchitis. Five daily intravenous injections of 1 c.gm. perchloride of mercury were given. The temperature fell by lysis to normal in six days and patient rapidly improved. (*See* Fig. 8.)

9. Case No. 183.—Treatment begun on the fourth day of illness. Temperature, 100°·4 F. Broncho-pneumonia was present. Five daily injections were given of 1 c.gm. perchloride of mercury. The temperature rose to 103° F. in the evening after the first two injections, and then gradually settled to normal. (*See* Fig. 9.)

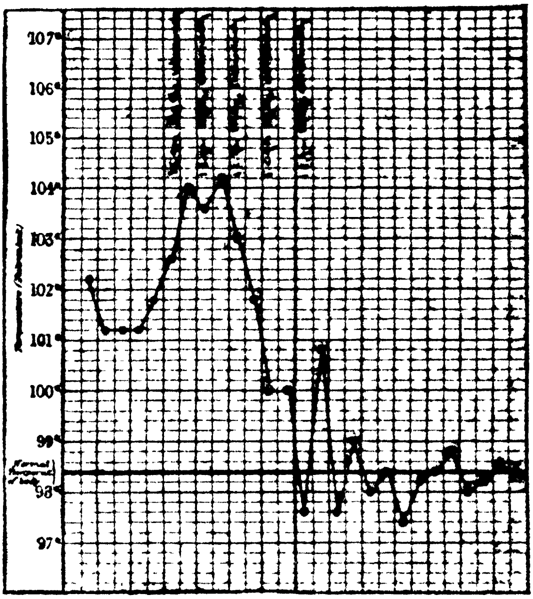

Fig. 7.

10. Case No. 181.—This is the last and most disappointing case of the series. Treatment was begun on the fourth day of the disease, with temperature 103°·4. and only signs of slight bronchitis in lungs and only slight signs of toxicity. Despite five daily injections of 1 c.gm. of perchloride of mercury, intravenously, bilateral broncho-pneumonia developed and the patient became desperately ill. On the thirteenth day of the disease, with temperature 102°·4 F., respirations, 44; and pulse, 154;

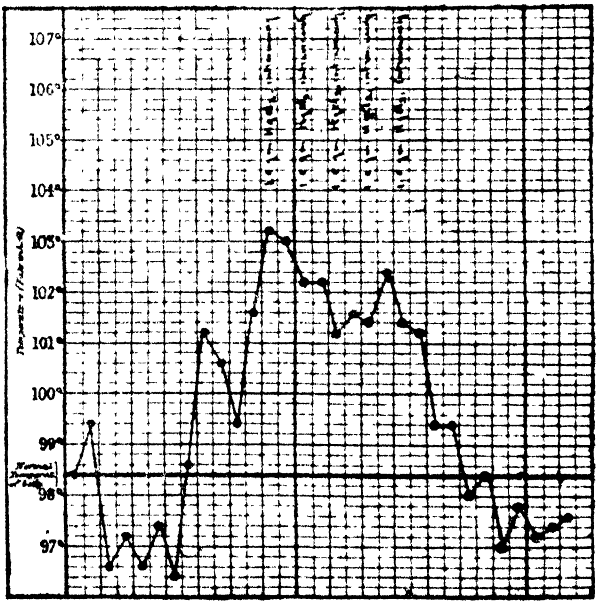

Fig 8.

600 cubic centimetres of 2·5 per cent. eusol were given intravenously. The patient appeared rather better the next day, but died the day but one later. (*See* Fig. 10.)

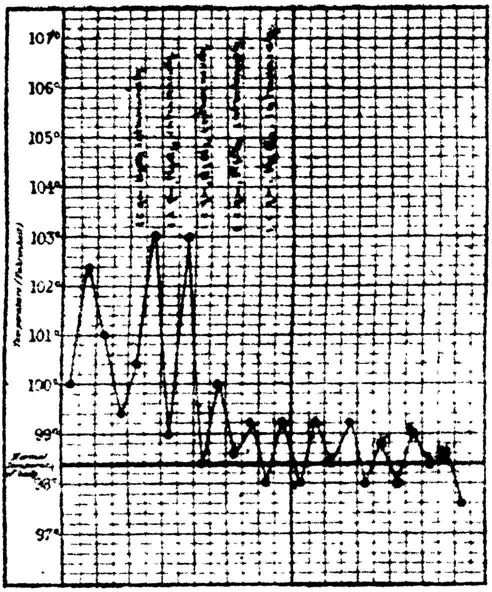

FIG. 9.

It seems that this is a fair test case. If intravenous injection of perchloride of mercury in these doses is really efficacious, it should have succeeded in this case, which was taken early, before broncho-pneumonia or even moderate bronchitis developed.

Since then the treatment has not been continued. The mortality in the series was 20 per cent.

BLOOD TRANSFUSION.

This was performed on one case (No. 90). He was desperately ill, cyanosed, and delirious. On the twelfth day of disease the temperature was 102° F. and bronchitis and broncho-pneumonia were present. He was classified, and found to belong to Class IV. Major D C. Taylor, M.C., R.A.M.C., operated on him, and removed first 450 cc. of blood from a vein in his arm; he then transfused him with 900 cc. of blood taken from a vein in the arm of a Class IV. blood donor. His colour improved and his delirium became less, but he died two days later. At the autopsy the liver was found congested, and the right side of the

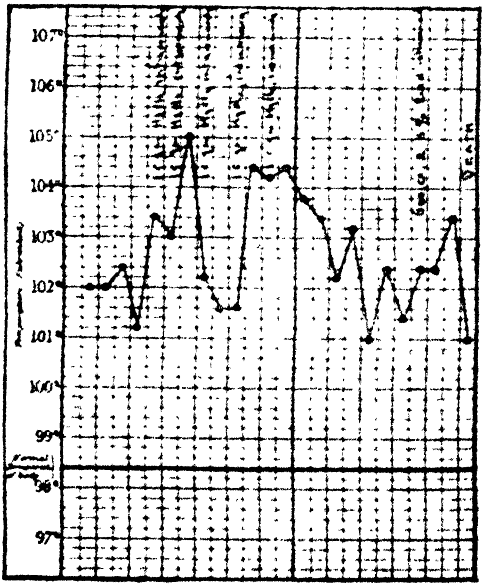

FIG. 10.

heart dilated.

FURTHER ACCESSORY MODES OF TREATMENT.

1. *Continuous oxygen* was administered to one patient (Case No. 89) by the Haldane apparatus for nearly 36 hours. He was almost in extremis, with double broncho-pneumonia and mottled cyanosis of the leaden type. The rate of respiration was lowered from 60 a minute to 50 a minute, but he did not survive. It merely seemed a method of prolonging the agony of death.

2. *Bleeding.*—Although at post-mortem examinations the right side of the heart is found dilated in a certain number of cases, it is rarely found to be much enlarged during life, as judged by physical examination. Bleeding, by venesection, has been done in a fair number of cases, but I have never seen any benefit result from it, and the blood is often extremely difficult to obtain in quantities exceeding a few ounces.

CONCLUSIONS AS TO TREATMENT.

1. None of the specific methods tried has proved satisfactory in every case.

2. The temperature chart does not afford a reliable criterion of the efficacy of any special form of treatment. Patients who have had no drugs, beyond aperients, will sometimes exhibit charts as striking as those seen after the administration of vaccines, salicin, or perchloride of mercury.

3. In the present state of our knowledge, symptomatic treatment alone is available.

REFERENCES.

1 Wynn: *Lancet*, December 28, 1918. **2** Turner: *B.M.J.*, August 3, 1918. **3** Pollard: *B.M.J.*, October., 26, 1918. **4** Turner: *B.M.J.*, March 8, 1919. **5** Beaumont: *Lancet*, August 9, 1919. **6** Siciliano: *Med. Supplement to the Review of the Foreign Press.* February 1919. **7** Ferrarini: *Riforma med.*, 1918, 34, 893–4. **8** Humbert: *Rev. méd. de la Suisse rom.*, Genève, 1918, 38, 713.

Splenomegaly and Jaundice–Splenectomy.

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THE diseases in which splenomegaly is associated with jaundice are linked together by many common clinical features, and perchance have some fundamental ætiological relationship.

The present article is an attempt to present the diagnostic features of these diseases, and to indicate the principles on which their treatment must depend.

The case that suggested the subject is that of a girl of 18, who was admitted to the Whitworth Hospital under Dr. Nesbitt's care in June, 1918. She came to hospital on account of a yellowish discoloration of the skin and daily attacks of severe pain in the right hypochondrium.

With the exception of measles at the age of seven, the patient had been healthy until 1916, when she first noticed the yellowness of the skin. This persisted for eight months, when she experienced a stabbing pain in the right hypochondrium followed in a few days by the development of a deeper yellow tint. During the next two months she had almost daily attacks of pain and the jaundice remained the same. A year after the onset of symptoms she entered another hospital, where she was operated on for gall-stones. None were found, but a cholecystostomy was established. The jaundice was not affected by the operation but the pain was relieved for some weeks, its return apparently synchronizing with the closure of the operative biliary fistula. Eight months after the operation the pain became more pronounced, starting in the region of the gall-bladder and radiating to the abdomen and chest. It lasted about five minutes and reappeared on the following days. At this time the jaundice deepened. She vomited seven or eight times in a fortnight, the vomitus being stained with bright red blood. She stated that she suffered from flatulence, and that her face had become rather swollen. Family history—negative.

On admission, the patient was a well-built and well-nourished irl, moderately jaundiced, the tongue was slightly furred, appetite good, and she had no distaste for fatty foods. She had occasional attacks of flatulence, but no nausea or vomiting in hospital. Bowels were normal, except for occasional diarrhœal attacks. Stools were clay-coloured on admission, but were normally coloured on all further examinations. The liver was not enlarged, there was tenderness on deep pressure in the region of the gall-bladder, where signs of the former operation were evident. The spleen extended about three inches from the left costal margin. There was no itching or pruritis. Heart sounds were normal, the pulse full, regular and easily compressible, its rate averaging 88. Blood examination June, 1918, showed hæmoglobin 80 per cent., red blood-corpuscles 6,000,000, leucocytes 8,000. Urine was highly coloured, and contained bile pigments. There was some frequency of micturition due to polyuria. The temperature was normal until July 20, when she had an attack of pyrexia without pain lasting for two days. During this attack of pyrexia the patient felt ill, but no definite change in her condition resulted.

During the six months in the Whitworth Hospital the patient had four attacks of malaise and abdominal pain, after which the jaundice deepened, but it always subsided to the moderate yellow tint again. On one occasion the presence of ascites was demonstrated.

On August 20, blood examination showed Hb. 80 per cent., rbcs. 4,500,000, leucocytes 4,000. On December 17, the Hb. was 80 per cent., the red cells were 4,000,000 the leucocytes 2,000, whilst a month later (on January 22, 1919) hæmoglobin had risen to 90 per cent., the red cells to 4,850,000, and the leucocytes to 5,625 per cm.

The differential count at this last examination was: polymorphonculears 66 per cent., lymphocytes 30 per cent., transitional 3 per cent., mast cells 1 per cent.

The urine, which contained bile pigments on admission, on further examinations gave no or extremely slight reactions with nitric acid and iodine. On January 22, no bile pigment was present. The reaction for excess of urobilinogen with Erlich's solution of dimethyl-paramino-benzaldehyde and hydrochloric acid was strongly positive on repeated examinations.

The outstanding features of this case are: chronic jaundice, afebrile exacerbations, enlargement of the spleen, leucopenia, the absence of enlargement of the liver, and the intermittent presence of bile in the urine. The diseases which were considered in the diagnosis were: Hanot's hypertrophic biliary cirrhosis, Banti's disease, and hæmolytic jaundice.

Before discussing the diagnosis of this case a short description of hæmolytic jaundice is not unnecessary, because this disease has not yet met with general recognition.

The cardinal symptoms are chronic enlargement of the spleen with an acholuric non-obstructive jaundice, and anæmia, frequently paroxysmal in character, and varying in intensity. The red blood-corpuscles show a diminished resistance to hypotonic salt solution.

Vital staining shows an increase in the number of reticulated cells. The urine contains an excess of urobilin (Krumbhaar[1]).

There are two types of the disease, an acquired, first clearly described by Hayem[2] in 1898, and a familial or congenital, described two years later by Minkowski.[3]

The acquired form of the disease usually begins with a definite attack of illness, accompanied by anæmia, the erythrocytes falling sometimes to 1,000,000, whilst the jaundice is slight or moderate. In the congenital form the patients may have had chronic jaundice from childhood, being otherwise in perfect health, but the complete picture of the disease unfolds with the years. The familial type is apparently the more common, and its study introduces several points of diagnostic significance.

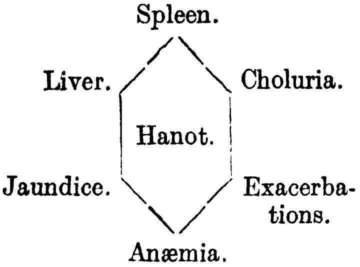
Elliott and Kanavel[4] describe three cases occurring in one family :—

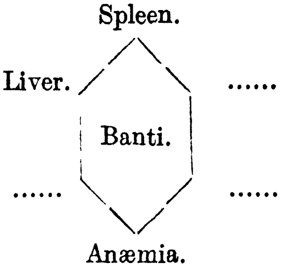
> The father, aged 54, showed all the symptoms of hæmolytic jaundice. His daughter, aged 22, presented essentially the same clinical picture without enlargement of the liver and without excess of urobilin. His son, aged 27, had had one attack of jaundice, the spleen was enlarged, the resistance of the red cells was slightly diminished while the blood-count was normal (red cells 5,184,000, white, 9,600, hæmoglobin 100 per cent.).

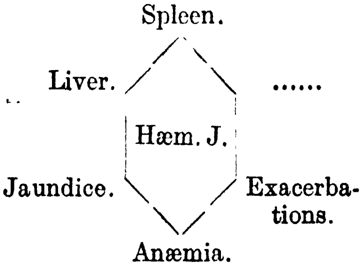
In the last patient, the enlargement of the spleen and the history of an attack of jaundice were the only positive features on ordinary examination. The fragility test added another suggestive indication. I mention this case to illustrate the fact that a normal blood-count and percentage of hæmoglobin is not incompatible with the diagnosis of hæmolytic jaundice.

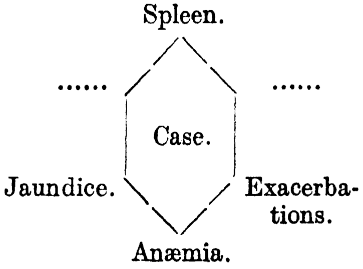
These family histories light up a case of apparently idiopathic splenomegaly with possibilities. The special methods of diagnosis include fragility tests and the examination of the urine for excess of urobilinogen and urobilin.

With regard to the diagnosis of my own case, I have tabulated the salient characteristics of Hanot's cirrhosis, Banti's disease, and hæmolytic jaundice as follows :—









It is not unusual for cases of Hanot's biliary cirrhosis to show signs of anæmia and for the spleen to be enlarged out of proportion to the enlargement of the liver, so that the similarity to Banti's disease may be striking, but it is decidedly uncommon to find no enlargement of the liver in Hanot's cirrhosis. The clinical resemblance between Hanot's cirrhosis and hæmolytic jaundice is more striking, as has been pointed out by Mayo.[5]

In both conditions young people are affected, the disease is of long duration, splenomegaly and hepatic enlargement may be present in both, jaundice is often slight and increasing after exacerbations of pain, ascites is usually absent, anæmia may be present in Hanot's cirrhosis, and in hæmolytic jaundice the blood-count may be normal.

Taking the clinical picture as a whole, we regarded this case as one of Hanot's cirrhosis in which the liver was not enlarged. We excluded Banti's disease on the presence of early and well-marked jaundice, the absence of ascites, and the absence of any definite hæmorrhages. Hæmolytic jaundice was excluded (in the absence of a fragility test), by the depth of the jaundice, by the leucopenia, and by the general clinical appearances of the case.

In presenting the clinical characteristics of these diseases schematically, it is not suggested that the diagnosis of any ailment can be accurately represented by the formula $a + b + c = x$, where a, b, etc. are prominent symptoms and x any given disease. Diagnosis depends on an accurate appreciation of the value of symptoms. In fact, the general appearance of the case is often of more significance than the summary of symptoms.

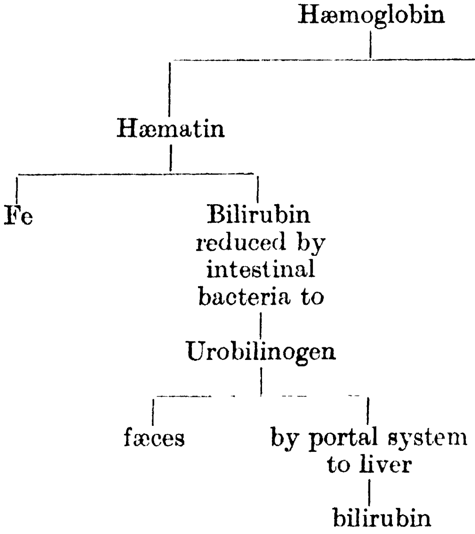
Such dogmatic representation, however, may serve to draw attention to outstanding features. It is obviously of first importance from a therapeutic standpoint whether a case comes under the category of hæmolytic jaundice or pernicious anæmia, under Banti's or Hanot's syndrome.

The existence of cases such as the one reported, and the definite clinical resemblance between Hanot's cirrhosis, Banti's disease, and hæmolytic jaundice suggest some relationship; though clinical resemblances do not necessarily imply ætiological identities.

No completely satisfactory hypothesis of the cause of these diseases has been advanced. There is one significant feature common to all, namely, that all three are accompanied by signs of increased blood destruction (Coppinger).

In all there is some degree of anæmia, an increased formation of bile pigments, and varying degrees of jaundice. The amount of urobilin and its precursor urobilinogen excreted seems to form the best index of the amount of blood destruction going on in the body.

I might represent the sequence of events thus—

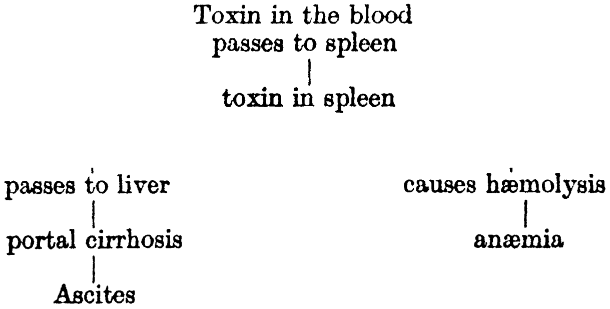

Hæmoglobin
Hæmatin
Globin
Fe
Bilirubin reduced by intestinal bacteria to
Urobilinogen
fæces
by portal system to liver
bilirubin

If we take it that bile pigments are derived solely from hæmoglobin, an excess of urobilinogen in the fæces indicates an increased disruption of the hæmoglobin molecule; an excess in the urine may indicate a deficiency of the liver cells in that they fail to convert urobilinogen into bilirubin, and the former is absorbed into the general circulation and appears as such in the urine.

The total excretion of urobilin in urine and fæces is increased according to several investigators in Hanot's cirrhosis, Banti's disease, and in hæmolytic jaundice. Hunter, in Albutt's system of medicine, states that urobilin is increased in the urine in fevers, pernicious anæmia, febrile forms of jaundice, and in cases in which absorption of blood is proceeding, in the first three of these conditions the functional activity of the liver is certainly impaired, and in the last urobilin is in all probability abstracted from the systemic blood by the kidneys without having gone to the liver at all. In old blood-clot, hæmatoidin is formed identical with bilirubin, of which urobilin is a reduction product identical with hydrobilirubin.

The excess of urobilinogen in my case simply points to a functional derangement of the liver, rather than to increased destruction of blood, but if the amount in the fæces had been estimated, we could have had an index of the amount of hæmolysis.

This evidence of increased destruction of the red blood corpuscles brings cases of Hanot's cirrhosis into line with those of Banti's disease and hæmolytic jaundice. As the theories of causation of the latter two affections have been more carefully formulated, I give them first, and I will then try to point out ætiological analogies. We may represent Banti's view of the sequence of events in the disease bearing his name thus:—

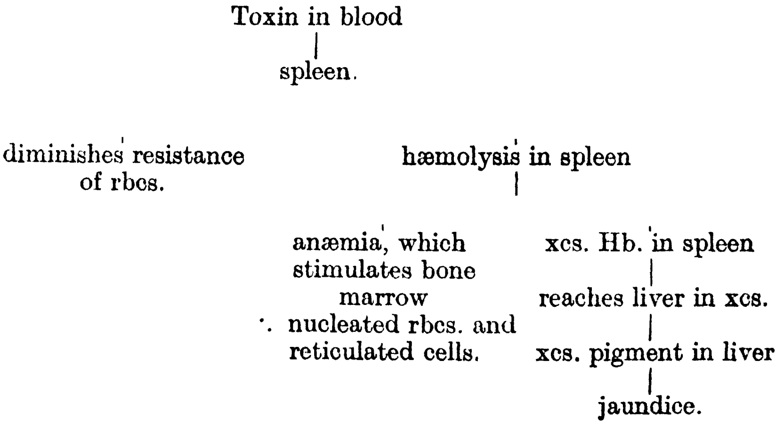


"The usual opinion is that the enlargement of the spleen is due to a chronic inflammatory process, which in turn results in an increased functional activity, *i.e.*, increased hæmolysis with a resultant anæmia" (Krumbhaar).

The increased urobilin elimination supports the opinion that the anæmia is due to hæmolysis. The decided improvement after splenectomy, and the histological evidence of the presence in the spleen of cells, which destroy red cells, point to the spleen as the cause and site of this destruction. One observation quoted by Krumbhaar indicates the *rôle* of the spleen in the causation of the secondary portal cirrhosis. Umber, during a splenectomy for Banti's disease, excised a piece of the enlarged liver for microscopical examination and found peripheral infiltration of the lobules. Later the liver returned to its normal size. This indicates that the removal of the spleen stopped the cirrhotic process in the liver.

Kidd[8] and others have reported definite disappearance of ascites after splenectomy in the later states of Banti's disease.

With regard to hæmolytic jaundice, the current theory is that the spleen destroys red blood cells and that it prepares others for destruction by diminishing their resistance. We might represent the process schematically, thus :—



In Banti's disease, the splenic toxin affects the liver to a larger extent, and the blood to a smaller, whereas in hæmolytic jaundice the reverse is the case, the liver being only involved in that it has to deal with an excess of hæmoglobin. We might presume a toxin, bacterial or otherwise, finding a nidus in the spleen, and either remaining there to act on the blood passing through that organ, or yielding a product or part of itself to the portal circulation thus setting up cirrhosis of the liver.

In other words, hæmolytic jaundice may be due to a toxin confined to the spleen, whereas Banti's disease may be due to a toxin most of which escapes to the liver.

Whatever the first cause of these diseases may be, it may be taken as proven that the spleen is an essential element in their causation, or in the determination of their activities.

We have considered the hæmolytic *rôle* of the spleen. We must now consider the manner in which the liver is involved in the pathological process. The effect on the liver of hæmolysis, and the consequent increased amount of hæmoglobin sent to it for excretion, may be stated thus:—

> "When free hæmoglobin is introduced into the circulation the liver takes it up and changes it into bile pigment which passes out through the bile passages in the usual manner. If, however, the hæmoglobin is taken up by the liver in larger quantities, and especially if this occurs rapidly, the pigment is formed faster than the bile capillaries can remove it, and it is reabsorbed into the circulation giving rise to jaundice and bile in the urine" (Krumbhaar).

Now, when hæmoglobin is set free in the portal circulation, the liver receives a more concentrated and rapid dose, and jaundice is more apt to occur than when the same amount is set free in the systemic circulation. The jaundice is proportional, not so much to the amount of hæmolysis but to the *site* of hæmolysis. The introduction of hæmoglobin into the general circulation, in doses which would produce jaundice in normal animals, fails to do so when the spleen is removed.

The explanation of these facts is found in the blood-supply of the liver. This organ receives 75 per cent. of its blood through the portal vein and 25 per cent. through the hepatic artery. It is clear that a considerable proportion of this 75 per cent. comes from the spleen. When, therefore, hæmoglobin is set free in the systemic circulation, much of it is diverted *en masse* to the portal system through the spleen, so that the liver receives a more concentrated dose than it would if the spleen were absent. These facts have significance in pointing out the possible part played by the spleen in Hanot's cirrhosis of the liver. This disease we assume to be due to a systemic toxin; as the liver is the only organ grossly affected, this toxin is apparently not injurious to other organs such as the kidneys (for there is no albuminuria and the casts found depend on the result of the hepatic lesion, *i.e.*, bile in the urine). The liver is involved because it is the only organ in the body which receives a double dose, and a concentrated dose of toxin from the spleen. If the spleen was cut out of the circulation, the liver would receive the toxin in a less concentrated form, and be able to deal with it as it can deal with hæmoglobin under similar circumstances. By this reasoning, the spleen is the mechanical means of determining the advent of larger doses of circulating toxins to the liver than are received by other organs.

The practical conclusion to be reached from these considerations is that, if there is any evidence that the liver is involved secondarily to the spleen, or if the spleen is a factor in the causation an adjuvant to the course of hepatic cirrhosis, splenectomy is indicated. The fact that the spleen is sometimes enlarged before the hepatic enlargement and before jaundice make their appearance, is usually taken as evidence of a systemic infection, but may signify a primary involvement of the spleen. The onset of a terminal portal cirrhosis is taken by most observers to indicate the passage of a toxin from the spleen.

I might sum up the reasons for splenectomy in cirrhosis of the liver:—

(1) It relieves the liver of work.

(2) It renders circulating toxins more dilute in the portal vein.

(3) It is of great or greater benefit in chronic conditions characterized by increased blood destruction.

(4) It has been productive in Mayo's hands of "extraordinarily good results."

I have only touched on the fringes of the problem of splenomegaly and jaundice; when we consider that recent experiments (Whipple[9]) seem to prove that bile-pigment may be derived from other sources than hæmoglobin, and that the liver is *not* the only site of its formation (McNee, Whipple[10]), but that bilirubin can be formed by the endothelial cells of the body generally, notably those of the spleen, kidney, and bone marrow, we realize that the first cause of jaundice may lie not in the liver but in other organs, and that it may be possible to protect the most important metabolic organ in the body by removing the spleen.

This paper represents in part the path to splenectomy along which I adventured—

Operation.—Splenectomy was performed and did not present any special difficulties. The patient did very well for 36 hours, when she suddenly vomited a large amount of blood. Her pulse became imperceptible. Twelve house later she vomited more blood, and died shortly afterwards.

Post-mortem report showed the stomach and duodenum full of blood without any lesion whatever of their walls. This gastric hæmorrhage was undoubtedly the cause of death and was in all probability due to the advanced cirrhotic state of the liver.

Pathological report on the liver is as follows:—"There are thick connective-tissue bands running through the section. These contain many newly-formed bile-ducts. In some areas the liver cells and their nuclei fail to stain. There is much bile staining of the cells and some of the bile-ducts are full of bile. I consider the liver a multi-lobular cirrhosis of the usual atrophic type

The spleen shows no alteration other than thickening of the walls of the venous sinuses in the pulp. There is also one epithelioid-celled tubercle in a Malphigian body." Both pleural cavities showed widespread adhesive pleurisy. The heart showed fatty degeneration.

There was no gross obstruction in the biliary tract, nor evidence of infection of the pancreas.

The microscopic appearances of the liver detailed above suggested to one pathologist portal cirrhosis and to another biliary cirrhosis of an infective type, but the histological changes of Hanot's cirrhosis were absent.

Taking the clinical and pathological evidence together the diagnosis must be biliary cirrhosis without any qualifying epithet.

For classification purposes, this case was obscure in life and in death was not clarified. It is reported at length because it seems important to supply data from which in time some conclusions may be drawn. Splenectomy was carried out deliberately for cirrhosis of the liver and not for any of the conditions for which that operation is usually performed.

If my theoretical discussion leads to a correct conclusion, splenectomy should be performed in early cases of cirrhosis of the liver, when it would certainly promise more than in the late case reported.

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Encephalitis Lethargica.

By ALFRED HOWELL, M.D., M.R.C.P.
Assistant Physician to King Edward VII. Hospital, Cardiff, etc.

ENCEPHALITIS LETHARGICA is a recently described infectious disease, with important and interesting features. Its incidence is considerable. During January, 1919, 103 cases were notified; during February, 116; and by the end of June, 1919, the total reached 351. During the warmer months—July, August, and September—there were only 64 cases. I am indebted to Dr. Walford, M.O.H., Cardiff, for these figures, which relate to England and Wales. The mortality is a serious one; of 126 cases collected by the L.G.B. in 1918, the deaths numbered 25, a mortality of 20 per cent.

The Registrar-General has kindly supplied me with the number of deaths registered in England and Wales, as from January 1, 1919 (when the disease became notifiable). During the first quarter the number was 128; during the second, 58; and during the third quarter, 39.

It will be seen that while 351 cases were notified for the half year, the deaths registered during the same period numbered 186, and indicate a considerably higher death rate than that observed in 1918. Netter, in France, had 7 deaths out of 15 cases; Economo, in Austria, 6 out of 11; of 10 cases seen by me, 5 died.

A feature of interest is the fact that the stress of infection falls largely on the mesencephalon. Pathological changes are found scattered throughout the brain, their intensity is greatest in the mid-brain.

One set of symptoms associated with mid-brain changes is that known as the extra-pyramidal symptom complex. Paresis, tremor, rigidity, are among its chief features; they are also (with stupor) the leading clinical signs in encephalitis lethargica. MacNalty connects the stupor with involvement of the afferent paths in the crus cerebri, and consequent blockage of the incoming stimuli.

Historical.—Cases were first noticed in Vienna, at the beginning of 1917. Netter published an account of cases in France on March 22, 1918. It was on April 20, 1918,[1] that two important papers dealing with the disease appeared; one by Dr. Wilfred Harris, and the other by Dr. Hall.

In view of the difficult, and in many ways defective, supply of food in this country, during the early period of 1918, the question of the possible connection between bad food and the new epidemic, had to be seriously considered by the authorities. Arrangements were immediately made by the Local Government Board and the Medical Research Committee for a thorough enquiry into the whole subject. By May 18, 108 cases had been reported. The Medical Officers to the Board issued a valuable and comprehensive report in 1918.[3] The main conclusions reached were as follows:—

1. That the disease was not caused by infection conveyed by the eating of food, good or bad.
2. In epidemics of infantile paralysis, a few of the cases had shown symptoms much like those of encephalitis lethargica cases described under the term polio-encephalitis. No connection was found between encephalitis lethargica and polio-encephalitis, or with the common type, poliomyelitis.
3. That the disease was *sui generis*, of an infective nature, identical with the encephalitis

lethargica of Economo and with the epidemic encephalitis of Netter.

The *clinical features* have been clearly dealt with[3] by McNalty in a study of 126 cases reported to the L.G.B. My own observations extend to 10 cases.

CLINICAL ÆTIOLOGY.

(a) *Age.*—There is no particular stress against any period of life. It is remarkable that the incidence, as regards age groups, is almost identical with that of the proportion of the whole population living at that age period. For instance, the number of people under 10 years forms 20·9 per cent. of the whole population. The percentage of cases of encephalitis lethargica occurring during that same age period is 20·6. For the second decade the figures are identical, viz., 19·0. For the third decade they are 15·8 and 17·3.

In my own short series of 10 cases, the distribution corresponds closely to that given above. This is in decided contrast to polio-myelitis, in which the percentages for the corresponding decades are 57·1, 22·1, and 9·0. Some observers have thought that the disease was a form of infantile paralysis, but this distribution would alone render such a theory very unlikely. (*See* table below published by L.G.B.)

	Enc. Lethargica.	Census Population, 1911. Proportion living at each Age Period.	Polio-myelitis.	Present Series.
Under 10 years -	20·6	20·9	57·1	1
10 to 19 - -	19·0	19·0	22·1	3
20 to 29 - -	15·8	17·3	9·0	1
30 to 39 - -	16·5	15·2	7·8	2
40 to 49 - -	13·4	11·5	4·0	1
50 to 59 - -	10·0	8·0	0	1
60 to 70 - -	4·7	5·1	0	1
70 years and over -	0	3·0	0	—

(b) *Sex.*—Of 225 deaths registered, 103 were males and 122 females. Of my cases, 3 were males and 7 females.

(c) *Season.*—The colder the weather, the greater is the number of cases. This is shown in the figures already given. On the other hand, infantile paralysis is notoriously a disease of warm weather, and especially of the later summer months. Of my own cases, six occurred in the first quarter of 1919 and four in the last quarter.

The *incubation period* is probably 10 to 14 days.

The *prodromal period* is indefinite and variable, and may last for a few hours or as long as three weeks. The chief symptoms during this time are headache, drowsiness, giddiness, general asthenia, irritability or depression, pains all over the body, and, occasionally, stiffness of the neck.

The *onset* again shows great variety. Lassitude, headache, and drowsiness are common. In three of my cases gastro-intestinal symptoms were pronounced —vomiting and severe diarrhœa. In another, pains in the joints, especially in the shoulders and knees, together with a raised temperature, suggested rheumatic fever. In a girl of 16, it began with twitching of the right side of the face and rotation of the head to the same side. Initial conjunctivitis is described as being common, occasionally there is tonsillitis. There was sore throat in only one of these cases.

Remissions during the earlier stages were observed more than once. Thus a soldier, the day after his discharge from the Army, developed pains " all over," with fever and lassitude, was ill for about 10 days, got better, went to work as a blacksmith for three weeks, and had to give up on account of increasing weakness; stupor developed rapidly and lasted three weeks, with a fatal result.

The *established disease* again presents a variable picture. The cardinal features are :—

Stupor.—It is the most characteristic feature of the complaint. It is present in a great majority of all cases. In my list every case showed it. It begins with apathy and may pass through drowsiness, lethargy, and deepen to coma. In an early stage, the patient may fall asleep at his work. The shortest duration that I have seen is three days, the longest three weeks. It has been known to persist for eight weeks. When fully developed the patient lies helpless, " like a log." The eyes are closed, the lines of the face obliterated, and the aspect is that seen in " Parkinson's Mask," a typical feature in paralysis agitans. On moving the limbs, there is a certain stiffness, much like the " lead-pipe " rigidity again observed in Parkinson's disease.

Katalonia may result when this rigidity is extreme, the limbs remaining in any position in which they are placed, as in catalepsy.

Two features deserve special emphasis. The first is that the day drowsiness may, and frequently does, alternate with nocturnal delirium. The second is that the patient can at times be wakened up from a profound stupor, and give a rational answer to questions.

Pyrexia.—This is observed in the great majority of cases. The temperature reaches 101° or 102°, rarely 103° to 104°. It lasts from 4 to 14 days. Complications like broncho-pneumonia and pleurisy are heralded by a secondary rise in temperature.

Asthenia.—Is constant and progressive.

Vomiting and *Diarrhœa* may occur in the prodromal stage, hardly ever in the established disease. On the contrary, persistent constipation is the rule.

Involvement of Cranial Nerves is frequent.—The third nerve is most frequently involved; the facial coming next in order, and more rarely the fourth and sixth nerves; and very rarely the hypoglossal. One of my series had a well-marked nasal voice.

Ptosis is the most usual feature, then squint with diplopia. Nystagmus is common, and is said to appear late in the disease. In one of my fatal cases it first occurred on the fifteenth and last day of the illness, spontaneous, horizontal, and persisted almost to the end, although the patient was comatose. These paralyses are usually slow in development, often bilateral, frequently transient. They usually clear up entirely. Paralysis is not limited to the cranial nerves; in one of my cases paresis of both lower limbs appeared three days after the onset, with some difficulty of speech.

Tremors.—Involuntary tremors are common, the arms being more usually affected. They may be violent and have been likened to those of chorea. Like the latter, they are exaggerated by exertion or excitement. One of my patients—a boy of 18—had irregular movements of his upper limbs, so severe that he had to be held down.

Reflexes present no characteristic features. The plantars are nearly always present; very rarely is an extensor (toe) response obtained.

Trophic.—Wasting may be extreme, and most definite in cases with signs of polyneuritis. No fibrillary tremors were seen.

I can find no reference to bed-sores in this disease. One of my patients developed a dusky infiltrated patch over the lower sacrum, the size of a saucer, two days before death.

Sphincters.—Retention of urine occurred in only one case; the catheter had to be used. It may be followed by incontinence, that is retention with overflow. Incontinence of urine is usual in the course of the disease, and is so common, running hand-in-hand with stupor, that these two symptoms occurring together in a doubtful case should always suggest the possibility of encephalitis lethargica as the cause.

Skin Rashes.—Of McNalty's 126 collected cases,[3] they were found in 22. Many kinds have been met with—erythematous, petechial, morbilliform, or scarlatiniform.

One of my cases, a girl eight years of age, developed a well-marked "scarlet" rash, all over the body, on the tenth day of her illness. There had been no preliminary sore throat, vomiting or rise of temperature. The rash disappeared in 24 hours. Thirteen days later, peeling, strictly limited to the soles of the feet, was noticed. Free general desquamation has been described. Two of my cases developed jaundice.

The *cerebro-spinal fluid* shows no definite changes, except that chlorides are present in excess. This is referred to again under "Diagnosis."

DIAGNOSIS.

The cardinal signs of encephalitis lethargica are pyrexia, stupor, asthenia, and cranial nerve palsy. Incontinence of urine is common, vomiting is rare, constipation the rule. There is no optic neuritis. There are no characteristic changes in the cerebro-spinal fluid. Cases with profound constitutional symptoms occur without any nerve lesions, and their fatality is great.

(a) *Tuberculous Meningitis.*—All the cardinal symptoms of encephalitis lethargica may occur in tuberculous meningitis. The early diagnosis of tuberculous meningitis is one of the most puzzling problems in medicine; at this stage tubercle bacilli are usually absent from the cerebro-spinal fluid, and may not be found throughout the whole course of the disease. The following points of difference deserve notice.

1. *Age Incidence.*—Tuberculous meningitis is most frequent in the first and second years of life, and rare after the tenth year. In encephalitis lethargica, 40 per cent. of all cases occur after the tenth year and, as already stated, its distribution is wonderfully

uniform throughout the whole life period.

2. *Vomiting.*—Common in tuberculous meningitis, rare in encephalitis.

3. *Reflexes.*—Brodzenski's reflex is present in tuberculous meningitis, absent in encephalitis.

[This reflex, also known as the "nuchal sign," is elicited as follows: The patient lies flat on his back, with lower limbs extended, the right hand is laid on the patient's chest to steady it, the left being used to flex the head on the thorax. If present, the lower limbs are immediately drawn up by flexion at the hip and knee joints.]

Differences in degree of response between the two sides as regards any one reflex are common in tuberculous meningitis.

Of 35 pairs of reflexes observed in encephalitis lethargica, I found definite difference between two sides only on two occasions.

4. *Cerebro-spinal Fluid.*—If T. bacilli are present, the diagnosis can be made at once. Chemical examination is of the greatest help.

	Normal.	Tuberc. Meningitis.	Enceph. Lethargica.
Albumen	·18	1·0 to 2·0 (infants) 2·0 to 3·0 (adults).	·22
Chlorides	7·32	5·0 to 6–0	8·56
Sugar	·53	Subnormal or absent	Normal.
Ash	8·8	7·5	8·7

The above table shows the proportions of the various constituents found in normal fluid and in that from cases of tuberculous meningitis as given by Mestrezat.[2] All the quantities are in grammes per litre. While investigating this question under the ægis of the Medical Research Committee, I have been able, in the main, to support these results. I have only been able to examine the fluid from two cases of encephalitis lethargica. Both were severe cases, one dying on the fifteenth and the other on the eighteenth day of the disease. Lumbar puncture was carried out on the fifteenth and thirteenth days respectively. The mean of the two results is given in the last column. The figures speak for themselves.

It is remarkable that while the chlorides in tuberculous meningitis are much below the normal, in encephalitis lethargica they are considerably above it. Incidentally, it may be mentioned that the figures given above for tuberculous meningitis are peculiar to that disease, and have therefore a corresponding value in diagnosis in any doubtful case.

(b) *Acute Polio-encephalitis* (or the cerebral form of acute polio-myelitis).—These cases are very rare, and have been described during an epidemic of the much more common polio-myelitis. In polio-encephalitis stupor is rare while convulsions are common. Further, in the same disease the paralysis is maximum from the outset, and usually noticed when the acute symptoms have disappeared. Draper[3] found in some of the cases reported as encephalitis lethargica cases with a residual palsy of a lower motor neurone type, like that seen in polio-myelitis. He considers such cases to have been almost certainly true cases of polio-myelitis.

(c) *Hysteria.*—Cases in which the irregular movements of the limbs are so violent as to necessitate forcible restraint, or the stuporous condition with katalonia, may be mistaken for hysteria. Paralysis of cranial nerves, incontinence of urine, and nystagmus are not found in hysteria, the deep reflexes are never lost, but the plantars are often absent. I found the plantars present in every case of encephalitis examined.

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On Syringing Ears.

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EARS may be syringed for diagnosis or for treatment, and, in either case, pus or wax may need removal. The process is not so simple as it sounds, and is one in the accomplishment of which mistakes are often made. A few words on the subject are not then out of place, and it would be well if our students definitely learnt and practised this surgical procedure, instead of taking it for granted that they know how to use it.

Two rules may be laid down. First, that water at the body temperature must be used; secondly, that for wax the syringing must be done hard, and for pus it must be done softly. To each of these rules there is an exception: to the first when the observer wishes to produce giddiness to test the function of the vestibular nerve; to the second when the patient has earache, and may have an acute otitis media, or other painful lesion, beneath the plug of wax.

To remove pus from the ear is easy, force is not needed and must not be used; an ounce or two of water will suffice to clear the ear, and any syringe will do. Even the glass syringe with a worsted plunger will provide the necessary flow, and being cheap is a convenient article for the patient to purchase if daily syringing at home is prescribed.

To remove wax from the ear may be a most difficult task; it needs great gentleness, and may need very great patience on the part of the surgeon, as well as the most consummate handling of his patient. The great thing to remember is to syringe hard. For this a good syringe is necessary, and the chief cause of failure is an inefficient syringe. It is most remarkable how many inefficient syringes there are on the market in the various instrument-makers' shops, and how many there are in the various wards and departments of the institutions of this country. The only explanation for this must be that sufficient attention has not been, and is not, paid to this elementary surgical procedure. Were it otherwise the trade many years ago would surely have changed the type of syringe that it sells. The best is a syringe which holds about 100 cc., and has a ground-glass barrel with a metal plunger; halfway along the plunger is a groove around its circumference into which a spring of wire fits; the plunger may be dipped in warm sterile vaseline, and will then fit the barrel without any leakage. Each end fits on to the barrel by bayonet joints, and the nozzle joins the distal end by a similar joint. Thus there is no chance of leakage throughout the syringe.

The criticisms which are raised to such a syringe are that it is easily broken, and that its price is prohibitive. The first criticism does not hold good, and the second should not. The writer knows of a syringe which answers the above description that was bought in London from Windler, of Berlin, during the International Congress of Medicine of 1913. During the war its owner took it in a field ambulance to three fronts. It travelled by wheel, pack-mule, and camel transport, and is still unbroken. In May, 1919, a leading firm of instrument makers was asked to make a similar syringe; the copy was delivered in December, 1919, and the price was four guineas. This was a nominal price, and was reputed to fall considerably short of what it had cost the firm to make the syringe. The chief source of difficulty seems to be that English glass factors do not stock the barrelling, and are so independent that they are not prepared to make it specially.

Failing such a syringe, one similar in all respects, but with the ground glass barrel replaced by a metal one, is best. This, however, has the disadvantage that one cannot see when there is air in the syringe and, as the plunger does not fit so accurately, water may escape into the proximal part of the barrel and be shot up on the sleeve and hand of the manipulator when the syringe is being refilled. Next in descending order of value come the syringes usually seen: syringes with screw joints which soon leak, syringes with leather washers which all leak, syringes with barrels that fit too tight, syringes with barrels that fit so loosely that they suck down more air than they suck up water, until the limit is reached in the syringe with a leather plunger that will not take to pieces at all. The nozzle will vary according to the idiosyncrasy of the user, some like a bulbous end, and some a straight end; a nozzle with a bend like an old-fashioned bayonet has many advantages.

The other common cause of failure and mistakes is the neglect to use the frontal mirror and the speculum. Often a patient will say that when he had his ears syringed, nothing came out, and on examination a normal empty external auditory meatus is seen. Or the patient says he had his ears syringed, that much wax was removed, but that since the syringing he has been very deaf, and in such a case the deeper wax is found still to be present softened and swollen by the moisture. A plug of hard dry wax may cause little or no deafness, but when part of this is removed the remainder takes up moisture and completely fills the lumen of the meatus, thus preventing the sound-waves reaching the drum. Such cases do not tend to make the person who has done the syringing popular with the patient, who sometimes needs much persuasion before he will believe that actual harm has not been done. All such cases would be obviated by the use of the frontal mirror. Nor need it be thought that this means that only specialists can syringe ears. It does not need much acquaintance with an aural speculum to see whether the ear is empty of wax or not. It is not necessary to say whether the drum-head is normal or not, and the necessary skill can be acquired by an intelligent nurse.

During syringing the wax has a tendency to come away either in little flakes or in one solid mass. The former is the case with soft wax and is the more troublesome, it may need several examinations and returns to the syringe before the meatus is empty. The latter occurs with hard wax, and may need much syringing, but when the solid chunk comes away it is nearly always in one piece and leaves the meatus quite clear and empty. When the plug of wax cannot at first be shifted, it may help to press the wax at its periphery away from the skin of the meatus with a view to making a channel for the water to get beneath the wax. With this exception the writer does not advocate the use of wax hooks, forceps, or other implements for the routine removal of wax. They are not so efficient, are more uncomfortable to the patient, and in the long run take up more time than does the syringe. It is a great temptation to take hold of a small piece of wax that looks as though it could easily be picked out, but each time it is touched the instrument tends to pass through without gripping, or the wax tends to slip away, and in the end resort must be had to syringing, after the patient has become resistant to manipulation.

These instruments have their uses in removing small flakes of epithelium which obstruct the view, or little spots of wax adherent to the wall of the meatus, but should never be considered to be the normal implement for use. This is the syringe.

If these precautions are taken it is seldom or never necessary to defer a patient for a second visit before the wax is removed. Some slight trouble to the surgeon may be saved by ordering drops of bicarbonate of soda in saturated solution, or of olive oil, wherewith to soften the wax, but if there is need for removal at one sitting it can be done.

One type of ear which is most difficult to get clean, is that with soft wax present in quantities not large enough to obstruct the lumen, but sufficient to narrow it and to stick to the end of the speculum as it is inserted; with this is a débris of moist epithelium softened and half-liquefied, so that it looks like pus, with which it may be mixed. Syringing with water leaves some sticking to the fine hairs at the opening of the auditory meatus, and the view remains obstructed. Such ears can best be cleaned by using a mop of wool soaked in liquid paraffin, which is a solvent of fat. This removes the epithelial débris and inspissated pus, and brushes down the small hairs so that they lie flat. It may be used, too, for ears full of sand and dust when these are causing a dermatitis. The writer first used petrol for this purpose and for long it worked well, but one day it seemed to hurt a small girl on whom it had been tried. He had his own ear filled with petrol, and after emptying it was able to confirm the statement of the girl. A delightful coolness was followed by a pleasing sensation of warmth and this by a burning which went on to a fiery heat, so that for half-an-hour he was walking about in agony unable to control the pain, to think, or to pay attention to any other subject. The pain appears to come when the drum-head is touched with the fluid, and it is suggested that the rapid evaporation of the petrol causes a freezing of the drum-head followed by a rapid thawing. This is not likely to occur with the liquid paraffin, which does not evaporate so rapidly.

The cleaning of ears is harder in children, and most difficult of all in that case in which it is most important, namely, the child with earache. To puncture the drum at the right moment may save the mastoid from becoming inflamed, to decide as to puncture, the meatus must be cleansed. The utmost gentleness is needed in every manipulation; perhaps a little wax can be removed with the syringe, a little more with forceps, some with the paraffin mop, and the last will come away with a further use of the syringe. One rule only can be laid down, that of patience and gentleness.

To complete our description, we may touch on the vestibular caloric test. This depends upon the fact that a current of the endolymph is set up by syringing the ear with water at a temperature different from that of the body. When such a current is set up there results a complex of symptoms known as giddiness or labyrinthine vertigo. One of these symptoms is nystagmus. When cold water is used, the nystagmus is set up away from the ear which is being syringed; in that case the vestibular nerve is not destroyed. The syringing must be done very gently, the object being merely to place the water in contact with the drum-head, so there is no need for more than a trickle. The water must not differ much from the body temperature; if it does, the unpleasant symptoms of the giddiness, nausea, and sensations of objects turning will arise cause the patient unnecessary discomfort.

Maladies and Medicines.

By ARTHUR CAMPBELL, B.A., M.D.
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I HAVE been led by two recent occurrences to jot down some few instances in which more or less ordinary cases or groups of cases have presented difficulties to me and to tell of the medicines which, personally, I have found most effective in their treatment. The late Sir Lauder Brunton, in a preface to Dr. Thomas Bodley Scott's *Modern Medicine*, says, "If other men will follow Dr. Scott's example and write down the results of their experience, the Medical Profession will benefit greatly by improvement in treatment." This encourages me in the selection of what may appear to be trivialities, but for what they are worth I present them.

For many reasons I think it not inexpedient to deal here with a few cases which present more or less unusual symptoms or were difficult to treat, and more particularly to mention the treatment which to me seemed most effective. I do not pretend that others have not come to the same conclusions, or that they may not have even better means of treatment for the diseases; I simply wish to pass on the methods I have found effective, and if they give satisfactory results to others my object will be realized. We all know that the same medicines for the same diseases administered by different doctors have not the same effects—what psychological influence lies behind this curious fact is beyond my intelligence to explain.

The first ailments I will mention are those acute or subacute respiratory diseases, with which we are all familiar: bronchial catarrh, bronchitis, pneumonia, and whooping cough. Emetine has been lauded for the early treatment, I presume, of these diseases, and, I think, rightly so. I have now had 40 years' experience, and I must confess that during the first half-dozen years of that period I tried many ways of dealing with the early stages of these diseases particularly, with very indifferent success and little satisfaction to myself.

It must be remembered that that was at a time when linseed-meal poultices were considered a *sine qua non* in the treatment, whilst steam kettles and ill-ventilated rooms, often cold and damp, increased my difficulties; moreover, any innovation introduced by a young doctor was a danger to his reputation and, therefore, a very risky thing to attempt, especially as during those years I was managing a branch for an elderly doctor, and my sense of responsibility to him perhaps exceeded reasonableness. In those days, acute respiratory diseases were wont to be treated with carbonate of ammonia, squill, and small doses of wine of ipecacuanha; possibly many are so treated even now in the early stages. The patient was either weighted down with a heavy poultice or chilled with a light, watery one; the windows were kept religiously closed, if not nailed up, and a fire in a bedroom was in most cases considered outrageous.

In the case of children, I found the routine treatment absolutely dangerous. Squill I found, as stated in one or two books, to be a narcotic irritant poison, which even in minute doses in children produced occasionally the following signs: congested face, pin-hole pupils, and unconsciousness; an emetic, however, always cleared up these conditions rapidly. I found that carbonate of ammonia produced a dryness of the mucous membrane, a condition quite the opposite of that we wished to obtain in the early stages, whilst ipecacuanha wine, although more or less suitable, produced, in adults particularly, too much nausea or sickness for the victim's comfort. After many trials I found the U.S.P. syrup of ipecac., with small doses of solution of bismuth, given frequently in the afternoon and evening and sparingly early in the day, to be the most quickly effective remedy in softening the harsh, dry cough, relieving the chest pain, and in reducing temperature and discomfort. To children 10 m. doses of the syrup may be given in very acute cases in the evening every ¼ or ½ hour until six or eight doses have been taken, and then at longer intervals. I debarred poulticing, persuaded the patient to have fires and ventilation, and, when the tongue was coated, gave a small dose of calomel. I also found that in adults, when there was much gastric disturbance, accompanying acute bronchitis, an effervescing mixture of bismuth in most cases effected a speedier recovery than any other form of treatment; when there is sickness, there is little of the dry, harsh cough. In the later stages, nux vomica, especially in pneumonia both of children and adults, is indicated, but my remarks are intended to refer to the early stages particularly.

The suggested emetine treatment of daily injections I have not yet tried; in country practice, the administration of hypodermics at regular intervals often presents difficulties. The constitution of ipecacuanha and its various preparations are interesting, and I will refer to them later.

As regards whooping cough, my practice is to keep the patient indoors until the cough has ceased; open windows, warm air, freedom from rapid air currents, are essential to rapid recovery, and I give freely the syrup of ipecac. and bismuth solution until the cough is quite soft and the whoop abated.

I have found, in obstinate neglected cases, Bordet's bacillus with pneumococcus, hypodermically, very effective; the injection is almost painless, and need only be done once a week for a few weeks.

And now to consider ipecacuanha.

Ipecacuanha is obtained from the dried root of Psychotria ipecacuanha (Rubiacea) or Minas ipecacuanha from Brazil; there is also a variety grown in the Straits Settlements, and another variety known in commerce as Carthagena I.

It was brought to Europe in 1648 and was used by an Anglo-Indian physician in 1858. Docker introduced large doses, 60 grs., twice or thrice daily, for severe dysentery in the Mauritius. Dr. Foy says we are indebted to Dr. Bardsley of Manchester, in 1829, for the use of emetine in dysentery, so the alkaloid is not of recent date.

There are three alkaloids found in the root: cæphiline, psychotrine, and emetine, as well as ipecacuanhic acid.

Briefly, emetine is prepared by extracting powdered ipecac. by alcohol, and from the extract obtaining the mixed alkaloids. Purification consists of careful elimination of cæphiline and psychotrine. Cæphiline hydrobromide crystallizes with great difficulty, while emetine hydrobromide is easily crystallized and separated.

As regards the treatment by emetine of amœbic dysentery and hepatitis, tuberculous hæmoptysis, cholera, and intestinal hæmorrhage in typhoid, I have no experience. I have tried the drug in mucous colitis; it had the effect I expected, it increased the diarrhœa. The drug is also recommended in hæmophilia and pyorrhœa alveolaris.

The relation between exophthalmic goitre and myxœdema seems now to be fairly clear; I allude to these ailments on account of several, to me, interesting points; first, Robert Graves was a relation of mine, although his description of exophthalmic goitre was given before I was born.

Secondly, the first case of myxœdema I saw was about the year 1883, a few days before the disease was described :—

I met a gentleman in my village very frequently, but had no opportunity of examining him; he presented the hairless face and head, the heavy swollen features, the slow speech and movements characteristic of the disease. Meeting his medical man, who was also at this seaside village for a spell, I asked what was the matter with the patient. He told me that there was absolutely nothing amiss, that he had seen many consultants and all agreed that he was free from ailment. Three days later myxœdema was described; on reading it I promptly wrote to my friend and colleague, but as up to that time no remedy had been found, the result was inevitable.

After a very prolonged and severe attack of exophthalmic goitre a patient of mine developed swellings on the legs indistinguishable from myxœdematous patches, which I have no doubt they were. I did not, however, test by using thyroid gland because other complications were more pressing at the time. These swellings cleared up after some time, but they must be rare sequelæ to this disease. Lately swellings of the face about the eyes were shown in an illustrated article in one of our periodicals, but as these occurred during an acute attack one would hardy expect thyroid to affect them favourably.

I will now mention a peculiar case of that sub-variety known as Charcot's latent exophthalmic goitre :—

There were two men in the same village known as "Crazy Smith" and "Lazy Smith." The first exhibited those peculiarities which were attributable to his nickname, the second had at intervals periods of inaction caused by those severe attacks of trembling, tachycardia (and in his case exophthalmos) which are characteristic of the disease and indistinguishable from many cases of shell shock we see now-a-days. This man had the peculiar feeling or knowledge of the date of an attack days and even weeks beforehand. For instance, he would call at my surgery, receive attention, and then ask when I would like to see him again. I would perhaps reply, "Come this day fortnight," his reply would be "Yes," or, "I am sorry I cannot come that day for my next attack will be on the day in question, but I will come the day before." Now on many occasions I visited him on the days named by him and with but one exception I invariably found him in bed, with severe tachycardia, trembling and an anxious pained expression betraying his sufferings. He died later on of phthisis, a mode of escaping from this world which, I believe, is not an uncommon termination to this disease.

I lately had a conversation with a prominent Londoner, who, although a layman, was much interested in "shell-shock," and was ardent in his views that "chess" if properly introduced to patients would tend to direct their minds and assist in curing. I need not say the suggestion came from one who admired the game and played it well, but I promised to find whether some cases were benefited or otherwise and report to him, so that any information on the subject would be welcome.

And now I will mention briefly epilepsy, a recrudescence of which disease is, I think, a notable feature of this war. I simply wish to mention the treatment because so few of the doctors I have met seem to have administered borax as a remedy. Borax in 20 gr. doses dissolved in hot water *ter die* is, I believe, the most effective treatment we at present are acquainted with; it is also very inexpensive, does not dull the intellect like bromides, is practically tasteless, and digestion is not interfered with by its use. I have tried many drugs in this disease, even veronal, but if veronal is useless in epilepsy it is of incalculable value in the convulsions of children, 2 or 3 grains dissolved in hot water for a child 6–18 months has never failed to relieve, and while I have never seen any bad result, I must add that probably owing to better methods of feeding and general care, convulsions in children are much rarer than in my early days of practice.

I saw my last case of puerperal eclampsia a few years ago. The patient had six convulsions within two hours; I was sent for and I administered 15 grains of veronal or barbitonum as it is now called. I managed to get the patient to swallow it during a lucid interval; she had one more convulsion a minute or so after taking the drug, but she had no other attack afterwards and made a quick recovery. In this case I also slowly injected into the rectum a considerable amount of normal saline solution, two methods of treatment directly in opposition to the views held by some—that veronal should never be given where there is any kidney weakness, and that the increase of fluid would be likely to waterlog the kidneys.

With apologies to the Ministry of Food, I should like to draw attention to the great advantage I have found in dieting my maternity cases; a reduction in the consumption of fat-producing foods makes an enormous difference to primiparæ and to those with deformed or narrowed pelves. I have no doubt many have advised patients in this matter, but to those who have not I would urge them to do so for the sake of their patients' welfare and for the saving of their own time and strength. Many are in town practices, and so cannot use a coarse argument so telling to a farmer's wife: "You don't fatten your sow when you expect her to farrow."

Another disease of rather rare occurrence, which has interested me considerably, is known as "nephritis dolorosa," the literature is very scanty, and the signs of the disease almost nil. Professor Casper recently wrote an article on the subject, which gives a very good picture of the disease. He mentions hot compresses and narcotics as remedies for the acute colicky stage, and the thermophore, mustard plasters, dry cupping, rest, a bland diet, pyramidon in small doses, aspirin and antipyrin for the later stages, but adds: "the effectiveness of all these remedies is questionable." A few years ago, chatting with Dr. Finlay, of Gloucester, about this nephritic colic, we had both found that our patients obtained prompt relief from repeated large doses of citrate of soda in warm water; dram doses in half a tumblerful of warm water every half-hour or hour for half-a-dozen doses never failed to give relief. The same drug in smaller doses twice or three times a day combined with an almost meat-free diet and abstention from much exercise gave excellent results after the acuteness of the attack had subsided.

Having stated that signs of the disease were very scanty, I may add that I have known it to be treated for several years as a heart affection in a case in which there was a perfectly healthy heart, but the sudden and excessive pain frequently caused faintness, and the affected kidney being on the left side the *fons et origo malis* was not discovered. In another case, in which proof of kidney affection was shown by the passage of a small calculus, an operation to cure the disease was performed on a foot distorted by old-standing infantile paralysis; I need not say the "cure" was not effective.

The disease, as described by Casper, is a "light glomerular nephritis"; it seems to me that, as there is usually considerable acidity of urine, there is a tendency to the formation of calculi. In some cases small stones are passed with relief to the patient, but in others no stone is formed, yet the severity of the pain in either case is very great, and permanent recovery has resulted in all cases I have seen up to the present.

Two diseases of the skin have yielded good results to certain forms of treatment, one of which, lichen, has been described recently as not being amenable to drug treatment. Urticaria was, I believe, the form alluded to; in the acute stages 15 or 20 gr. doses of bicarbonate of soda with 3 or 4 min. of liq. arsenicalis *ter die* almost invariably give speedy relief, and the itching is allayed by an ointment containing 60 grs. calomel and 40 grs. camphor to the ounce, the camphor being dissolved in a few minims of spirit. This ointment also quickly removes the silver scales of psoriasis, and, if continued, will not only relieve the irritation but in most cases cure the ailment for a time at least.

The experiences related will, I hope, assist others; if so, they will fulfil the purpose for which this article in THE PRACTITIONER has been written.

The "Ascaris Lumbricoides" as the Cause of Urgent Symptoms in Disease Amongst Children.

By CHARLES PENTLAND, M.D., M.Ch., B.A., J.P.

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Medical Officer of Health; Medical Attendant, Royal Irish
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In my practice amongst children during the past six months, I have been very much struck by the number of cases of severe acute forms of sickness directly attributable to worms, and practically almost in every case to the "ascaris lumbricoides." The embryo, as we are aware, forms in each ovum after its discharge in the stools, and then re-enters the body, by means, it is supposed, of drinking water, vegetables, or impure starchy material; but the endeavour to trace the exact cause of the appearance of worms in so many children is found somewhat difficult. In most of the cases which came under my notice, the source of the water was from "spring wells," samples of which I have had analysed. These were reported pure and free from traces of ova of the ascaris.

Contrary to what one would expect, all of the children before being attacked were of an unusually healthy and vigorous type; one is accustomed to associate worms chiefly with strumous and delicate children. An acute attack of worms presents, from a diagnostic point of view, symptoms which are by no means characteristic. I have found that severe pain referred to the umbilical region has been the most constant symptom, associated with a high temperature ranging from 105° to 103°. Closely simulating an attack of peritonitis or appendicitis in its early stage, the tenderness apparent in some of the cases has been very pronounced and most acute; other local symptoms have been uneasiness, attacks of severe colic, vomiting and faintness, capricious appetite, and irregularity of bowels, constipation and diarrhœa alternating. When one is confronted on a first visit with the following type of case, it is extremely difficult to form a diagnosis :—

Child, æt. 5 years.—Tongue heavily coated, of a putty-like appearance, great *malaise*, complaining of severe pain and tenderness in region of umbilicus, cough, and temperature 104°, with constipation, but no history of having passed worms according to the knowledge of the child's mother.

I have had three such typical cases during the past month, A., B., and C. :—

In each case I prescribed a diaphoretic mixture, and santonin and hydrarg. c. cret., with the result that, on the first evacuation of the bowels in Case A, five round worms, measuring from 4 to 6 inches, were expelled; in Case B, 12 worms were expelled, measuring from 2 to 4 inches; in Case C, one large worm measuring about 5 inches was vomited, and five small ones were expelled per rectum. In Case A, the temperature, which was 104°, came down to normal on the second day. In Case B, the temperature, which was 105°, was normal on fourth day, and in Case C, the temperature, which was never above 102°, came to normal on fifth day. No further symptoms were manifested, and the children appeared in normal health, quite lively, and anxious for food, though subsequently these cases showed a fair amount of anæmia which has practically disappeared with the administration of the usual tonics.

Another case equally remarkable and equally difficult of explanation occurred in my practice some months ago :—

Child, æt. 9 years.—Temperature 102°, tongue roughly coated, great thirst and a distinct degree of jaundice present, much tenderness over right hypogastrium, with vomiting and delirium. This case, however, had a history of having frequently passed worms a month previous to my seeing her. After the usual examination and excluding urgent diseases, I prescribed santonin powders combined with hydrarg. subchlor.; in two days afterwards a large round worm was evacuated. I continued the calomel in 1 grain doses for six days; the jaundice rapidly cleared up; three small worms were passed. About the twelfth day the patient returned to health quite normally, all symptoms of jaundice having disappeared.

Here, I am of opinion that the worm must have been lodged in the common bile duct, causing temporary jaundice. These cases may be of interest in so far as the symptoms appeared to be so unusual of worms, and resembling more the forms of acute disease. In the many forms of sickness, no matter how rare, amongst children it is advisable not to overlook worms as a probable cause of the urgent and distressing symptoms which one so often meets with. In the treatment of worms amongst children, it is essential to build up the general condition, and improve it by administration of tonics, cod liver oil, etc., as well as to clear out thoroughly the alimentary canal by keeping the action of the bowels free in order to avoid unhealthy accumulation of mucus. Important prophylactic measures, militating against the development of worms in children, are the preservation of the digestive organs, strict cleanliness, fresh air, and attention to oral hygiene. Stools known to contain worms or their ova should be burned immediately.

Practical Notes.

The Sachs-Georgi Test for Syphilis.

Galli-Valerio points out that although the Wassermann test may be excellent for the diagnosis of syphilis, its technique is so delicate and complicated, that slight faults in carrying it out may lead to false results. This explains why the same serum examined in different laboratories so often gives such varying results. He insists on the necessity for a test, which, if less exact than the Wassermann, has the advantage of being much less liable to errors of technique. The method by precipitation is certainly the most simple and the most practical, but unfortunately its results are not at all good. He recommends the method, described by Sachs and Georgi in 1918, by means of precipitation or flocculation, which he believes will replace, in most cases, the use of the Wassermann. It is based upon the fact that a mixture of syphilitic serum and an extract of organs containing cholesterin, gives rise to the formation of flakes of globulin of more or less bulk, which settle down at the bottom of the test tube. The extract is prepared in the following way: 100 cc. of alcoholic extract of bullocks heart (1 g. of heart to 5 cc. of alcohol), 200 cc. of alcohol and 13·5 cc. of a 1 per cent. alcoholic solution of cholesterine.

For use, 1 part of this extract is mixed with 1 part of normal saline (0·85 per cent.) and shaken up gently; and then 4 parts of the saline are added quickly. This yields an opalescent fluid, which must be used at once for the reaction. It is important that only solutions of the strength mentioned, 0·85 per cent., should be used, because any other strength will give either wrong results, or none at all. It must be clean, fresh, and sterile.

The serum must be quite fresh and clear, and should be inactivated by exposure to a temperature of 55°–60° C. for half an hour.

The technique is quite simple. To 1 cc. of the patients' serum, diluted 10 times with the normal saline solution, is added 0·5 cc. of the extract diluted as already pointed out. After being well mixed, the solution is placed in the incubator at 37° C., and remains there for 2 hours. It is then removed and kept at room-temperature for from 20 to 48 hours, and the result noted. After 48 hours the result is vitiated by the appearance of false precipitates. According to Meyer the result can be obtained more quickly by keeping the tubes in the incubator for 3 to 4 hours, and then putting them in the centrifuge.

A control is made at the same time with normal serum and the saline solution mixed in the same way with the cholesterin extract. The results can be read best by inclining the tubes against the black table of a dissecting microscope and examining them with a No. 8 lens (Leitz).

The results obtained by many observers show that only a very small percentage fail to correspond to a positive Wassermann, and it is considered by Lesser to be more reliable than the Wassermann.—(*Correspond. bl. für Schweizer Aertze,* December 25, 1919.)

Treatment of Dyspepsia in Biliary Lithiasis.

For the relief of the dyspeptic symptoms, acid eructations, pyloric spasm, adhesions round the pylorus to the liver, and pain in the stomach, Lœper recommends small but frequent meals consisting of pulpy food and very little to drink. The gastric sensations can be relieved by taking—

Cocaine hydrochlorate - - -	2 cg.
Lime water - - - -	100 g.

One dessert spoonful one quarter of an hour before the meal.

Tincture of belladonna, Tincture of hyoscyamus - - of each	3 g.
Aethereal tincture of valerian - -	10 g.

15 drops in a little water one quarter of an hour before the meal.

For pain coming on after the meal, morphia, codeia, and belladonna are useful, but better is dionine in a dose of 1 cg. For those who cannot swallow drugs easily, a suppository should be ordered.

Extract of valerian - - -	0·08 g.
Extract of belladonna - - -	0·02 g.
Dionine - - - - - -	0·02 g.
Cacao butter - - - - -	3·0 g.

Vichy water is often badly tolerated, and even aggravates the pain when taken as treatment of the lithiasis. In such cases it should be replaced by

Hydrated magnesia, Bismuth subnitrate - - of each	1 g.
Sulphate of sodium - - -	0·5 g.
Powdered sugar - - - - -	3 g.

To be taken half an hour before the morning and evening meals in a little Vittel water.—(*Journ. des Praticiens,* January 17, 1920.)

Reviews of Books.

Notes on Galvanism and Faradism. Second edition. By E. M. MAGILL, M.B., B.Sc., D.P.H., R.C.S.I. Pp. xvi + 224. London: H. K. Lewis & Co. Ltd. 6s. net.

IT is a very difficult matter to write an elementary book. It needs a very thorough knowledge of the subject, as it must be free from mistakes; and yet it is very difficult for the possessor of much knowledge in a subject to appreciate the standpoint of a beginner. The work under review is written with the avowed object of preparing masseuses for examination, and for this purpose it is doubtless of value. No doubt, a striving for brevity makes its descriptions rather uninteresting in some instances. On the other hand, a certain amount of elementary electricity, which is chiefly of historical interest and not applicable to medicine, might be cut out. The description of the Wimshurst machine is weak, and no mention is made in it of inductance. Another point that should be eliminated is the suggestion that nurses or masseuses should use a dangerous drug such as aconitine for ionic medication: the author must have forgotten for the moment that the drug is introduced into the blood eventually in any ionic medication, and administration of the poisonous alkaloids should only be made under the strictest medical supervision. However, the book condenses a great deal into a comparatively small space.

Baby Welfare. By W. E. ROBINSON, M.D. Pp. 206. London: T. Fisher Unwin. 7s. 6d. net.

THIS small volume presents in a few pages the bulk of what the general practitioner must know in order to run a baby-welfare department successfully. As there must be a great increase in this type of work in the near future, there would seem to be likely to be a considerable demand for such a book, and although there is, perhaps, rather more dogmatism than will be to the taste of some readers, these will at least be stimulated to observe and possibly to record their disagreement. On the whole, we can cordially recommend any man who is embarking on the care of a baby welfare department to read this book as a preliminary to his work. He will acquire some fresh ideas and will be able to test the truth of some of the statements made.

The Problem of Sex Diseases. By A. CORBET-SMITH, Barrister-at-Law. Pp. 107. London: J. Bale, Sons and Danielsson, Ltd. 2s. 6d. net.

ALTHOUGH the book is somewhat luridly painted, and rarities are made to appear as if of daily occurrence, a fault to be found in all the propaganda work on venereal diseases, which frighten, and often do more harm than good, there is no doubt that this volume will appeal to many and benefit some. A few inaccuracies appear, but the book is well written and in many respects the subject-matter is better dealt with than could be done by a medical man. Much may be done to lessen venereal disease by teaching the individual, but we fear it will not go so far as the author anticipates, and even the prophylactic measures now in use have fallen very short of what was expected of them. The author also falls into the error of overestimating the curative value of the present-day remedies. The author is so obviously anxious to do all he can to help in combating the scourge that we recommend the book to be read, but in the spirit in which it was written.

Criminology. By MAURICE PARMELEE, Ph.D. Pp. 522. New York: The Macmillan Company.

IT is difficult in a limited space to give any adequate review of this closely written book, which deals in detail with the wide subject of criminology. The work is divided into six parts, viz., 1: Nature and Evolution of Crime; 2: Criminogenic factors in the environment; 3: Criminal traits and types; 4: Criminal Jurisprudence; 5: Penology; 6: Crime and Social progress. It will thus be seen what a wide field has been covered, and while all the sections will be found instructive to those interested in the subject, the section on punishment will, perhaps, appeal as much as any to the ordinary reader who follows the general social problems of the day.

Post-mortem Manual: A Handbook of Morbid Anatomy and Post-mortem Technique. By CHARLES R. BOX, M.D., B.Sc., F.R.C.P., F.R.C.S. Pp. 372. London: J. and A. Churchill. 8s. 6d. net.

WE welcome the second edition of this little book, which is intended as a guide for students engaged in post-mortem work and medical men called upon in the course of practice to conduct autopsies. The making of a post-mortem examination for a coroner's inquest is a task which is generally avoided, if possible, by the general practitioner, for various reasons, which may include lack of familiarity with the process, fear of committing some error, and the possibility of making an unsatisfactory appearance in the witness box. With such a guide as this book, however, to refresh his memory, the general practitioner should be able to acquit himself creditably, and should find difficulty in none but exceptionally obscure cases. The methods of investigating the various regions and organs of the body are carefully described, and under each organ is included an account of the pathological appearances resulting from disease. The book is concisely written, yet remarkably complete in detail. We cordially recommend it.

Preparations, Inventions, Etc.

VITMAR.

(London: Messrs. Callard & Co., 74, Regent Street, W.1.)

This preparation is manufactured from wheat, bone-marrow, eggs, fruit, and sugar by a delicate process which ensures the presence in full activity of the three essential vitamines. The fat is completely emulsified, and so is made easily tolerable.

It is light yellow in colour and of syrupy consistency, having a sweet, fruity, and pleasant flavour and an agreeable aroma.

The presence of the vitamines in the preparation has been proved by biological experiments thoroughly carried out under strict conditions. For this reason it is of high value in the treatment of malnutrition, bad development, deficiency diseases, and in convalescence after severe and exhausting affections of all kinds.

ENERGEN DIABETIC ROLLS.

(London: Frank G. Hyman, 23, Thavies Inn, Holborn Circus, E.C.1.)

These rolls are very appetizing in appearance, and prove to be equally pleasant for eating, being crisp, easily masticated, and of good flavour.

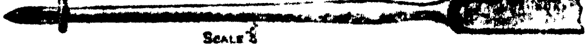
Energen is a product from fresh wheat gluten, has a high food value, and is digestible and assimilable.

The bread has been found, in many cases, an acceptable substitute for ordinary bread in the treatment of diabetes, obesity, indigestion and other disorders requiring a limitation of starchy food in the dietary.

A TONSIL-BAYONET.

(London: Messrs. Allen & Hanburys, Ltd., 48, Wigmore Street, W.1.)

Mr. Scott Gillett has devised this instrument for opening peritonsillar abscesses without the disadvantages attaching to the use of a scalpel guarded with strapping or of unguarded forceps. The advantages claimed for this instrument are that it is easily sterilized, that the presence of the collar prevents the surgeon from stabbing too deeply, and that the double edge of the bayonet makes it easy to convert the stab into an incision upwards or downwards as required, after which the operation can be completed with the of blunt forceps.



THE PRACTITIONER

MAY
1920

Trauma and Tuberculosis.

By SIR THOMAS OLIVER, M.D., F.R.C.P.
Professor of Practice of Medicine, University of Durham College of Medicine, Newcastle-upon-Tyne.

THE possible causal relationship of trauma and tuberculosis has for several years engaged the attention of the medical profession. The Workmen's Compensation Act has given fresh interest to the subject.

In THE PRACTITIONER for June, 1912, Dr. Arthur J. Hall, of Sheffield, contributed a helpful paper as to how far trauma might be a possible factor in the production of disease. The enquiry is beset with difficulties, for disease is frequently an entity of such a complex character that many circumstances contribute to its development, although only one or two apparent causes can be suspected. Modern pathology has increased the difficulty of correlating trauma and tuberculosis, for tuberculosis, being a specific disease and due to a particular microorganism, it is hardly possible to see how injury can give rise to such a definite malady. When, therefore, tuberculosis follows upon injury, it is more in consonance with pathological experience to believe that a tuberculous lesion had been present in the body previous to the accident, and had been lying dormant. All that injury, therefore, could do, would be to rouse into activity a lesion which has been inert, probably for years.

That tubercle bacilli can remain locked up in the body, and inactive for years, has for long been an accepted fact, so much so that there are many physicians who believe that when pulmonary tuberculosis appears rather unexpectedly in adults beyond the thirties, it is possibly due to a relighting of glandular or other focal lesions which had originated in childhood. In 1892 Pizzoni examined the lymphatic glands removed from 30 dead bodies. During life, not one of the individuals had given evidence of tuberculosis, and yet when emulsions made from these glands were injected into guinea-pigs, 42 per cent. of the animals developed tuberculosis. On the other hand, Dr. Louis Cobbett[1] states that in experiments made for the Royal Commission, emulsions taken from caseous lymphatic glands of children failed to induce tuberculosis when injected into guinea-pigs, a matter of surprise to him, since in the caseous glands numerous well-formed tubercle bacilli were readily visible under the microscope.

As similar experiments carried out by A. S. Griffith, Weber, Eastwood, and F. Griffith equally failed to develop tuberculosis, it would appear as if in a large percentage of old caseous glands tubercle bacilli are either dead or have lost their virulence, and are hardly likely to be reactivated. The results of a still larger number of experiments, carried out by many pathologists, lend weight to the view advanced by Pizzoni, the interesting point being that many of the glands appeared normal to the naked eye, and yet under the microscope they exhibited the histological changes characteristic of tubercle. Admitting, therefore, the latency of tuberculosis, it is easy to understand how readily disease might be reactivated by injury, as in a case reported by Broadbent, where a youth, in whose sputum tubercle bacilli had been found, and whose health had been restored, remained well for several years until after falling from a horse he developed acute tuberculosis, to which he succumbed in six weeks.

My first experience of the possible relationship of trauma and tuberculosis was in the case of a young woman, aged 17, who, when crossing a busy street in Newcastle, was knocked down by a passing vehicle. Until the time of the accident patient had been quite healthy. Her chest was bruised by the accident; no ribs were broken. In an hour or two after the injury she had rather a sharp hæmoptysis. From the day of the accident her health gradually declined. When I saw her three months afterwards she had then well-marked signs of phthisis, to which she succumbed in the course of a few weeks.

I was impressed by the circumstance at the time. Since then several cases of almost similar character have come under my observation, of which the following are examples:

A mason's labourer, who had never lost time through ill-health, fell from a scaffolding and injured his right chest by striking a brick wall. He rested for a short period, but thought little of the accident. At the close of the day, he walked to the railway station and trained home, a distance of four miles. Next morning he was at work as usual, and continued to follow his employment until the ninth day, when he had rather a sharp hæmoptysis. This subsided, and he returned to work. Six weeks after the injury the man was seized with acute pain in the right chest. Pleurisy developed; patient's condition gradually got worse; cough and expectoration increased, and emaciation became rapidly progressive. Tubercle bacilli were found in the sputum, and there were well-marked signs of phthisis in the apex of the right lung. The case was tried in the County Court. The Judge awarded full compensation on the ground that the phthisis to which the man succumbed was the result of the accident.

A coal miner, when emptying a waggon, fell over the side, bruising his chest against the grease-box. An hour after the injury he had hæmoptysis. Notwithstanding this, he went on with his work, sparing himself as much as he could. That evening he had a restless night. Next morning there was a recurrence of the bleeding, and this continued off and on for four days. Gradually cough developed attended by signs of cavitation of lungs. Tubercle bacilli were found in the sputum. This patient did well under open air treatment.

A farmer, aged 30, when working in Canada fell off a hay stack, a distance of 30 feet. He fractured a rib, the broken end of which punctured the lung. Hæmoptysis occurred shortly afterwards. He was removed into hospital, where he remained the greater part of a year owing to recurring pulmonary hæmorrhage. He recovered sufficiently to return to the Old Country, and leased a farm in the Isle of Wight. There he lived the active life of a farmer for five years, during which his health was good, but, later, hæmorrhage returned at intervals and he died from pulmonary phthisis seven years after the injury. On enquiry I found that the reason why patient had gone to Canada was that as a clerk he had shown signs of commencing pulmonary tuberculosis. In this case, therefore, there is little doubt that a tuberculous lesion in the lung pre-existed the injury.

Professor E. Terrile[2] details the case of a workman, 52 years of age, who by a fall injured his left foot. The foot rapidly became œdematous, and when patient was admitted into hospital his malady was diagnosed as arthro-synovitis. A cold abscess formed. This was opened and the cavity packed with iodoform; a fistula however remained. Subsequently he developed cough, and died from what was regarded as a fulminating form of pulmonary tuberculosis.

As tuberculosis in connection with bone is hardly ever a primary affection, and there is little chance of tubercle bacilli penetrating the unbroken skin even in injury, the alternative theory is that the bacilli came from a pre-existing focus in the glands or elsewhere. That is the view Borri and A. Broca also take of the matter, their opinion being that, when injury to a joint is followed by tuberculosis, trauma only activates latent tubercle. Louis taught that there is no tubercle in the less frequented sites of the body unless it is already pre-existing in the lungs.

The subject of trauma and tuberculosis has received additional interest and opportunities of investigation during the War, owing to the large number of gunshot wounds of the chest wall, superficial and penetrating. My own War Hospital experience is that only in an extremeley small number of wounded soldiers has the relationship been observed. Dënéschau[3] (d'Anger) gives the details of officers and soldiers of healthy ancestry whose health was excellent until in battle they received gunshot wounds of the chest.

In one of the patients it was merely a superficial wound, the lung having been untouched by shrapnel. The same evening patient had hæmoptysis; six months afterwards he was emaciated and the subject of cough attended by pyrexia and recurrent hæmorrhage. Eighteen months later there were well-marked signs of phthisis with indications of cavity, and in the sputum numerous tubercle bacilli were found.

Another young soldier had his lung pierced by a bullet and immediately afterwards he had pulmonary hæmorrhage. For a month off and on the sputum was streaked with blood. Having partially recovered he was sent to a convalescent camp, where, during the next three months he was never free from fever, and the lungs were the seat of catarrh. Tubercle bacilli were found in the expectoration.

In Deneschau's first patient phthisis followed upon an external injury to the chest wall, and in the second, after a penetrating wound of the lung. Deneschau describes other cases in which injuries to the chest wall were followed by pleurisy first on the wounded side and then on the other. Bilateral pleurisy with effusion in the absence of kidney, heart disease, and a history of rheumatism, is extremely suggestive of tuberculosis.

Space will not permit of me dealing with the question of tuberculosis having followed "gassing."

In injury to the chest wall and its consequences the three main events are: (1) injury, with or without immediate damage to the lung; (2) hæmoptysis; and (3) subsequent phthisis.

To the question: Does injury to the chest wall cause pulmonary hæmorrhage, an affirmative answer can be given from the cases I have reported. When phthisis follows, is the hæmoptysis responsible for the malady? in other words, is there *phthisis ab hæmoptæ?* I cannot, at the time of writing, lay my hand upon the reference, but I remember reading some years ago an account of the sequelæ which followed the intra-tracheal injection of blood into rabbits. In those animals which were kept in a warm and comparatively even temperature it was found that the blood corpuscles had become disintegrated and that the lungs exhibited no structural alteration, but it was different in those who had not been similarly protected. In these an alveolar catarrh had been induced, and this had become a suitable soil for the growth and reproduction of micro-organisms.

It is not quite clear how injury to the chest wall causes hæmoptysis. If a rib is broken and the sharp edge penetrates a lung, hæmorrhage is easily explained, but it is different where the ribs remain unbroken. When, in these cases, bleeding occurs immediately after an accident the hæmorrhage might be the result of direct injury to blood vessels as the result of shock, but in many instances the hæmoptysis does not occur until an hour or two, or it maybe a few days or weeks, after the injury. The fact of hæmorrhage not occurring until several days after the accident is of itself suggestive that there was probably a pre-existing tuberculous lesion in the lung.

The question I have raised is not simply one of trauma and tuberculosis of lungs, but of other organs as well, and although I have already alluded to affections of the joints, this part of the enquiry may be all the better if further amplified. Max Schuler injected human tuberculous expectoration into the lungs of dogs and rabbits; he then sprained or otherwise injured their knee-joints, with the result that in nearly all the animals there was produced a granular inflammation of the joints with structural changes in the cartilages and underlying bone, whereas in control healthy animals the same rough treatment provoked no lesion at all. It would seem, therefore, that in the infected animals tubercle bacilli had found their way into the injured joints.

There is one drawback to an experimentally produced morbid condition of animal body and the effect of injury, and it is, that the animals experimented upon are never quite in the same position as a man or woman who for years has retained within his or her lungs or glands a tuberculous lesion which had apparently not impaired health. We are, therefore, not surprised when Lannelongue and Achard tell us, as the results of their experiments upon guinea pigs, that although all the animals died from tuberculosis, in not one of them were tubercle bacilli found in the injured joints.

The results of Petrow's experiments, on the other hand, lend qualified support to the opinions expressed by Max Schuler. Petrow first injured the joints of animals, and then injected into them, as also into non-injured joints, tuberculous material. In non-injured joints the lesions which developed were limited to the ligaments and capsules, whereas in injured joints the continuity of the capsules was interrupted and had become a nucleus of tubercle. After having injected tuberculous material into 18 rabbits, Petrow induced 18 intracapsular fractures, 18 sprains, and 18 slight contusions. At the end of 3½ months all the animals had died. All of them showed signs of tuberculosis. Two of the slightly contused joints were tuberculous, also two of the joints which had not been subjected to injury. Injury, therefore, had only exercised slight influence in determining to a joint tubercle bacilli, when the tubercle micro-organism was already present in the body. Add to these data the facts drawn from our own experience. Time and again tuberculous patients receive injuries to their joints, and yet in how remarkably few instances is a tuberculous arthritis set up. If, then, it is difficult for an injury to incite tuberculosis of a joint in persons suffering from well-marked tuberculosis, how much more difficult it must be for a tuberculous joint to develop after injury in persons who at the time of the accident are believed to be healthy.

Applying the above to the suspected relationship of trauma of the chest wall and subsequent phthisis we may say that, as a consequence of injury to the thorax in persons who have a tuberculous focus in their lungs, laceration of these organs takes place attended by hæmorrhage into and around the diseased patch or patches, that tubercle bacilli, until then incarcerated, are liberated, and that these gaining entrance into the circulation find in their new surroundings renewed opportunity for reproduction. Sourdille, for example, reports the case of a man who died eight days after receiving a kick on the chest from a horse. No ribs were broken. At the autopsy a considerable quantity of sero-sanguinolent fluid was found in the left pleural cavity; there were also atelectasis of the lower lobe of the left lung, congestion of the upper lobe, and at the level of the injury a pouch which contained black clotted blood. The pouch, which was of the size of a duck's egg, lay just underneath the pleura and its walls were irregular. In addition, one bronchus had been torn across. Had this man lived for several months after the accident instead of a few days only, it is more than likely that the injured condition of the lungs would have proved an attractive and suitable soil for the reception and reproduction of micro-organisms, and especially of tubercle bacilli; if the latter, then presuming the lungs to have been previously healthy, it would have been a case of post-traumatic phthisis by infection.

Dealing, therefore, with the three events: (1) injury; (2) hæmoptysis; and (3) phthisis, it would at first sight appear as if the injury was the primary link in the chain of morbid events. But can we be quite sure? Knowing as we do that in at least 70 per cent. of our adult population there are dormant foci of tuberculosis, and that previous to the injury there were no signs of the latent pre-existing tuberculous lesions, is it not more likely that these constituted the more important link in the chain of events, and but for which traumatism could not have played the *rôle* it did in determining the morbid conditions which led to death? At any rate I confess my inability to see how *traumatism per se* can bring into existence a disease of the lungs so distinctly microbial as pulmonary tuberculosis, but I readily admit that it may light up disease which is quiescent by liberating micro-organisms held up in the lungs or elsewhere, and that these infect fresh portions of the lungs or are carried to distant parts of the body, or, as in the case reported by Sourdille, trauma by reducing local and general resistance creates a condition of soil which is favourable to the development of tubercle bacilli introduced from without. In one or other of the two last possibilities lies, in my opinion, the answer to the relationship of trauma and tuberculosis.

REFERENCES.

[1] *The Causes of Tuberculosis,* pp. 70–71, Cambridge Public Health Series, 1917.

[2] *Il Lavoro,* December 31, 1915.

[3] *Bull. et Mem. de la Société Médicale des Hôpitaux de Paris,* 27 juillet 1916, p. 1284.

Medical Notes.

(SECOND SERIES.)

BY SIR THOMAS HORDER, M.D., F.R.C.P.
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DISEASES OF THE LIVER.

IN palpation of the liver, it is well to adopt a definite method. Perhaps the following is as good as any:

(i) *Find the lower border and trace its outline.* In doing this, care should be taken to begin the palpation sufficiently low down to ensure not missing the edge of an organ that is larger than was anticipated. If this precaution is not adopted, and the surface of the liver happens to be smooth, it is not unlikely that the observer will report that the lower border cannot be felt. This error is avoided by beginning to palpate in the right iliac fossa and proceeding gradually upwards.

(ii) *Ascertain the characters of the lower border*—thin or thick, regular or irregular, normal consistency or hard, everted.

(iii) *Explore the anterior surface* of the organ for uniformity or lack of it, and for the number and size of any irregularities, with any special feature these may possess, *e.g.*, umbilication.

(iv) *Judge of the massiveness of the organ,* using the bimanual method, which has also been employed in (i) and (ii).

(v) *Judge also of the degree of fixation of the organ* by bimanual palpation when the patient is in the genu-pectoral position.

(111) There are three stages in the course of alcoholic cirrhosis of the liver: portal congestion, portal pressure, and cholæmia. In the first stage, the symptoms are chiefly those of chronic gastritis, and physical signs are, for the most part, absent. The symptoms and signs during the second stage are chiefly referable to the collateral portal circulation. The symptoms of the third stage are toxic, and are analogous to those met with in icterus gravis. But in most cases this third stage never arrives, because the patient succumbs to one or other of the serious complications of the second stage (hæmatemesis, portal thrombosis, heart failure), or to one of those infective processes to which the patient is specially liable (tuberculosis, streptococcus and pneumococcus infection).

(112) Hæmatemesis in cirrhosis of the liver may occur during the stage of portal congestion, in which case the bleeding is due to more or less general oozing from the congested gastric mucosa, and is not of serious consequence; or it may occur during the stage of portal pressure (collateral portal circulation), when the blood usually comes from an ulcerated varicose vein at the lower end of the œsophagus and is of very serious significance. If this distinction can be made clearly in any particular case, the treatment is considerably helped. In the early type of hæmorrhage, portal depletives may be used freely with advantage; in the latter type, the treatment should be on the lines adopted in the hæmatemesis of gastric ulcer.

(113) The state of compensation which is seen in many cases of cirrhosis of the liver during the second stage may be maintained at a fairly good level for

several years, provided the patient gives up all or most of his alcohol and adopts a simple dietary. This condition of things is rarely seen in hospital practice—a fact which explains the bad prognosis in respect of time given in this disease by many authors.

(114) The distinction which has been made between cirrhosis of the liver and perihepatitis in regard to ascites, namely, that when ascites recurs several times after paracentesis the pathological condition present is perihepatitis and not cirrhosis, is probably much too arbitrary. It is doubtless true that, *cæteris paribus*, perihepatitis is more constantly followed by ascites than is cirrhosis of the liver; but it is also true that recurring ascites not seldom complicates cirrhosis in the absence of perihepatitis.

(115) *Fever is common in cirrhosis of the liver, but the cause of it is by no means always apparent.* Serious causes that should be passed in review are pleural, pulmonary, and peritoneal tuberculosis, septic pylephlebitis, and cholecystitis. When the cause is obscure and the degree of pyrexia slight, the fever is probably related to subinfection of the peritoneum by micro-organisms of low virulence. Chemical and cytological examination of the ascitic fluid and post-mortem examination of the peritoneum yield evidence that chronic peritonitis is common in cirrhosis of the liver.

(*To be continued.*)

Medico-Legal Notes

(*continued*).

By SIR JOHN COLLIE, C.M.G., M.D., J.P.

Medical Examiner to the London County Council; late Director of Medical Services, Ministry of Pensions; Home Office Medical Referee, Workmen's Compensation Acts, etc.

MANY patients suffering from a traumatic neurosis suffer not so much from the physical effects of the injury, which has produced the condition, as from an exaggerated idea of the severity of the injury. Neurotic people who have been unfortunate enough to meet with an accident are, as a rule, much more in need of treatment than their traumatism. One of the most thorny problems in medico-legal cases is to determine how much of an existing condition is the effect of trauma and shock, and how much is due to a faulty nervous system or a perverted mental outlook.

Justly to estimate how far a patient's symptoms are attributable to his or her faulty mental processes entails an intimate knowledge of the symptoms of both neurasthenia and hysteria, and considerable practical experience in the treatment of these conditions. Generally speaking, it may be said the more an accident results in actual physical injury, such as a broken bone, the less likelihood there is of serious nervous *sequelæ*, especially if the case is wisely treated from the first. The European war gave innumerable instances of this. Those who treated casualties abroad, and those who had to deal with the men after they had recovered from their original injury, are well aware of this.

In cases in which the arbitration of Civil Courts has to be evoked, one sees innumerable instances in which the law's delay actually leads to the development of superadded functional nerve disease. Too often the keynote of the situation is the desire for a lump sum settlement.

It would be interesting to know how many cases of what was at one time known as Erichsen's railway spine or "concussion of the spine" (which, I take it, really meant concussion of the spinal cord), had they occurred in action in France would have been cured long before they reached the base; yet railway companies were for many years mulcted in large damages in respect of these cases.

Traumatic neurasthenia is a psychical condition, and is particularly intractable to any but intelligent treatment. Provided the physical disability, which has caused cessation from work, has ceased to operate, firmness and the *vis-a-tergo* method is the best, and often the only effectual, method of cure. Counter-suggestion should be immediate, forcible, and continued, and if applied at the psychological moment is marvellously successful. These conditions were recognized by the Army Medical Authorities, and the success with which functional nerve disease was treated in the field was one of the many bright spots of military medical treatment. When President of the Special Medical Board, I induced the British Red Cross Society and the Ministry of Pensions to open the First Home of Recovery in England for ex-soldiers suffering from neurasthenia, and subsequently, when I was Director of Medical Services to the Ministry of Pensions, a considerable number of similar institutions was opened throughout the country. When the need for these special institutions had been demonstrated, the Army Medical Department opened several for undischarged soldiers, and I believe the ground is now fairly well covered for this class of case.

Unfortunately, in civil life we have not the same control of the patient, nor have we opportunities for properly carrying out the treatment in its early stages. It is almost invariably useless to try and convince a working man, who has set his mind upon obtaining damages by means of an action at law, that a firm determination to turn a deaf ear to his perverted sensations and return to work, when he is able, is really in his best interests. So many coins of the realm appear to him of more value than self-respect and a return to work. It is curious to notice, as I have done in many cases, how soon the money so obtained is dissipated, and how quickly the plaintiff, having no further claim, returns to work when the money is spent.

A working man has, what he calls, "ricked" or sprained his back. What happens as a rule? No attempt is made to make an accurate diagnosis; indeed, there is no injury which is diagnosed so loosely, and none where the exact pathological condition engages less attention.

In dealing with alleged injury to the back there always comes a time, and the sooner the better, when one has to make up one's mind whether the pain is real, psychic, or assumed. The real difficulty, however, is not in determining to which category the case belongs, for there are very few cases of real injury to the back which are not accompanied by profound mental effect. Many comparatively slight injuries of this region are often grossly exaggerated, and too frequently are accompanied by conscious or unconscious malingering. It is well to remember that in the large majority of cases of alleged pain in the back the pain is psychic, not physical.

Working men who are subject to lumbago often allege that a sprain has induced the commencement of the attack. Dr. R. T. Taylor has aptly remarked: "Lumbago is pain and stiffness in the back not due to an accident. A sprained back is an accident with pain and stiffness, and the distinction is subtle." Lumbago is produced by an inflammation of the fibrous tissue surrounding the fusiform nerve-end organs which lie between the muscle fibres. This explains the extreme pain produced by movement. There is generally a rise of temperature during the first few days, but later it is impossible to tell, from clinical examination, whether an alleged injury is producing the pain complained of or whether the condition is one of an ordinary attack of lumbago, unless, indeed, there is external evidence of bruising and the condition is unilateral. Lumbago does not last long; pain following an injury may be indefinite.

Fortunately, cases of alleged pain in the back, which are likely to end in the Law Courts, are only sent to us for examination many weeks, and it may be months, after the accident.

All cases of alleged accident occurring to the numerous employees of the London County Council and the Metropolitan Water Board are sent to me almost at once, and in these one cannot assume that time has eliminated the presence of lumbago. The following illustrates the difficulties of this class of case when complicated with fraud:—

A. G., aged 41, was notified for examination in respect of an alleged accident while on duty, for which he had been on the sick list for 28 days. He said that whilst at work he felt something "give way" in his back. Admittedly he had had no medical attendance during the past 16 days. He reported himself unfit to visit me, and was examined at his own house. Later, although reported as fit to do so, he again refused to visit me when ordered, and declined to furnish a medical certificate as to his inability unless his employer paid for it. Finally, he was brought to my house in a cab.

Examination.—He was a healthy, powerful man, who showed no signs of disease. He complained of pain when touched anywhere. There was on his back the remains of a plaster. On the pretext of removing this, his back was vigorously scrubbed, and he made no complaint of tenderness. Later, although he said he could not stoop, I induced him to touch his toes on two occasions. Whilst his attention was taken up with the question whether the electric current from a battery was or was not flowing, he made no complaint of firm pressure with metal electrodes along the spine.

He was obviously deliberately malingering, and I certified him as fit to resume duty at once. He returned to work a few days later, did light work for two days, and then stopped. After a few days he resumed work at window-cleaning, but continued to attend the electrical department of an hospital. He was submitted to me for re-examination. I arranged for the presence at this consultation of the surgeon in charge of the electrical department of the hospital he was attending, who agreed with me that the pain in his loins the man now complained of was a pretence. When told he would have to go back to full duty, he at once agreed, saying he was "happy to do so." He evidently felt the game was up. This employee had deliberately malingered; his defiance of authority, and his readiness to take full duty when detected, proved him to be an undesirable servant. He was therefore dismissed.

Result.—He commenced arbitration proceedings against his employers, and the case was heard at the County Court four and a half months later, resulting in a decision in favour of the employers.

A few weeks subsequently I was examining one of his fellow-workmen, who volunteered the following interesting piece of information: that A. G. informed him, *three days prior to the alleged straining of his back,* that he was suffering from lumbago!

(*To be continued.*)

Vaccines in the Treatment of Cutaneous Diseases.

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EVERY new form of treatment tends to go through a phase in which its value is exaggerated and its employment experimental and unrestricted, and it is only after it has passed through this phase that its utility becomes properly estimated. Vaccine therapy has proved no exception to this rule, and may be said to be still going through the initial period; at the present time, it is being employed so carelessly and promiscuously and so often in cases in which it is so completely inappropriate, that there is a danger of its being discredited and its value unduly depreciated.

During the last few years vaccines have been used extensively in the treatment of skin diseases, and their effect can be gauged more accurately in this connection than in disease of any other organ, for improvement or aggravation resulting from their use may be detected so readily. In spite of this they are being used in many instances in a manner which borders on scientific quackery. It is no uncommon occurrence to meet with patients suffering from cutaneous diseases, such as psoriasis or Lichen planus, in which no causal organism has yet been isolated, who have been subjected to prolonged courses of vaccine treatment, sometimes unfortunately with no other object than the extraction of fees, and sometimes from a desire to retain a patient by trying a new form of treatment in a resistant case which had baffled all previous efforts at cure. It is equally common to encounter patients who are being treated with vaccines prescribed by someone who did not possess the requisite knowledge to diagnose correctly the skin affection he was treating, or by one who had sufficient skill to cultivate an organism from the disease and to make a vaccine from it but who had not taken the trouble to ascertain whether the organism cultivated was the true cause or simply a contamination.

In April, 1919, the *British Journal of Dermatology and Syphilis* published a series of short papers on vaccine therapy in diseases of the skin, by various physicians in charge of skin departments of the London hospitals who had given the treatment an extensive trial and had carried it out under the most favourable conditions with the collaboration of expert bacteriologists and properly equipped bacteriological laboratories. In view of the discrepancy of opinion on this subject which prevails at present, it may be of interest to the readers of THE PRACTITIONER to have a brief summary of the conclusions arrived at by these writers.

Dr. H. G. Adamson, St. Bartholomew's Hospital, expresses the following views: "My own experience in the treatment of sycosis, of pustular acne, and of other chronic staphylococcal infections by vaccines has been distinctly disappointing, and I am unable to relate a single case of this sort which has been cured, or of which I could say that it has been definitely benefited by the treatment. When I say this, I refer to chronic cases of sycosis, acne, furunculosis and staphylococcal impetigo. In recent cases of furunculosis the results have been somewhat more encouraging. In many of these more acute cases, cures have resulted apparently as the result of vaccine treatment, although even here it is difficult to lay down any rules as to dosage, or to know beforehand whether vaccines are or are not likely to do good. In the treatment of lupus vulgaris by tuberculin I have been altogether unfortunate, for although some cases have at first seemed to improve, there has been subsequently in nearly all a more rapid spread of the disease."

Professor Arthur Whitfield, King's College, considers that the following opinion will meet with little contradiction, namely, that "it is undoubtedly the fact that some cases have been so favourably influenced by the inoculation treatment that this favourable influence cannot be dismissed lightly as a mere coincidence of the treatment with a spontaneously developing favourable turn in the disease. Secondly, that admitting the previous statement to be true, many cases have shown no such favourable influence, and that, while only few may be said to have been definitely aggravated by the treatment, many seem to have been entirely unaltered."

He thus summarizes his views on the inoculation treatment:—

"(1) In certain diseases which usually run a short course under what I may call ordinary treatment, the inoculation treatment often achieves brilliant results in the exceptional cases where the malady proves resistant and tends to degenerate into a chronic malady.

"(2) In most cases of relapsing acute infections of the skin where no constitutional defect and no exposure to local irritants (other than bacteria, of course) can be detected, the inoculation treatment very frequently interrupts and brings to an end the series of relapses.

"(3) In diseases of clearly chronic course, and especially in those in which the skin undergoes obvious structural change, inoculation has in my hands proved unavailing, though I have carried it on in hospital patients for two years.

"(4) In dermatoses apparently dependent on infections of other parts of the body, but not themselves infective in nature, there is some evidence that inoculation treatment of the infected organ aids in restoring to the skin its normal power of resisting adverse external influences."

Dr. J. H. Sequeira, London Hospital, and Dr. G. T. Western, of the Inoculation Department of that hospital, after discussing some general points, detail the conditions in which vaccine therapy is frequently employed and record their experience:—

In staphylococcic infections, they had obtained their most striking successes in cases of deep-seated furuncle and carbuncle, but had been less fortunate in staphylococcic folliculitis. Coccogenic sycosis of the beard also proved resistant to the treatment except in early stages.

In streptococcic infections, striking results had often followed the use of a suitable streptococcic vaccine; in erysipelas it had caused a rapid fall of temperature and an abolition of the toxic condition.

In mixed infections, decided benefit had been obtained occasionally by vaccine therapy; a case is recorded in which an ulcer spreading from a colotomy wound involving one-half of the left side of the abdominal wall, in the discharge from which were found bacillus coli communis, bacillus pyocyaneus, saccharomyces, and staphylococcus aureus, healed with remarkable rapidity under staphylococcic vaccine. In tuberculous affections, the dry type of lupus vulgaris did not respond to vaccine therapy, but scrofulodermia and lupus of the ulcerative type had proved more amenable, and they had seen large ulcerated areas spreading from broken-down tuberculous glands heal up under it.

In gonorrhœal keratodermia, they had seen remarkable improvement follow the use of gonococcus vaccine, the carapace-like lesions on the soles having separated rapidly and the skin returned speedily to the normal condition.

In acne vulgaris, the vaccine treatment was frequently disappointing, and it was only in the pustular forms of that disease that they had met with occasional success.

In acne rosacea, since this condition depends on internal causes and is not primarily due to a local infection of the skin, they did not advocate the employment of vaccines in its treatment.

Dr. J. M. H. MacLeod, Charing Cross Hospital, and Dr. W. W. C. Topley, Director of the Pathological Institute of that hospital, came to the following conclusions:—

Of all the cutaneous conditions which they treated with vaccines, the only ones in which immediate and definite benefit resulted were suppurating staphylococcic lesions, especially acute recent and recurrent boils; by vaccines, both stock and autogenous, they had been able to cause the rapid involution of boils without the assistance of any form of local treatment and in almost every case to keep the patient free from recurrences, though there was often a tendency to relapse after the treatment was discontinued. In the case of chronic boils, such as those about theback of the neck, in which the circulation through the boil is impeded by the formation of scar tissue, the results were uncertain and, as a rule, unsatisfactory.

In acne vulgaris, the results were not encouraging; where suppurative lesions predominated, the vaccine treatment caused a diminution in the pustulation but did not influence the comedones, and when it was discontinued an exacerbation of suppuration generally took place. In those cases of acne, chiefly in adult women, in which the comedones were few or absent and which were associated with small, more or less indolent subcutaneous abscesses, little or no benefit was derived from the treatment.

In sycosis barbæ of coccogenic origin, the results were uncertain, and not to be compared with those from other methods.

In tuberculosis cutis, both the old tuberculin and bacillary emulsion were tried. Improvement was obtained from Koch's original tuberculin in lupus with superficially ulcerated patches, and healing had taken place in such lesions on the subsidence of the local reaction; but this procedure, even when small doses were given, was regarded as too dangerous for fear of lighting into activity unknown foci of the disease in other organs. The results with bacillary emulsion were irregular and, though some improvement was obtained at times in superficially ulcerated patches of lupus and in scrofulodermia, in no case did they find that the benefit from it was in any way equal to that which could be obtained by appropriate local treatment.

The Opaque Meal and the Opaque Enema.

THEIR DIAGNOSTIC POSSIBILITIES AND LIMITATIONS.

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AMONG medical men of all sorts—physicians, surgeons, and general practitioners—one hears the most diverse views expressed as to the value of the opaque meal in abdominal diagnosis. These views range from an almost complete scepticism to a pathetic belief that nearly every problem concerned with the viscera below the diaphragm can be solved by efficient X-ray investigation. Of these two attitudes, the former is by far the more dangerous so far as the public is concerned, for a competent radiologist is always careful to point out, in each individual case, the precise limits of his findings.

One frequently hears expressions such as "the X-rays revealed nothing" or "the X-rays showed an ulcer in the stomach." Statements of this kind are fundamentally wrong, for they fail to take note of the personal factor, which is all important. One man will discern much where another draws blank; on the other hand, misinterpretation of shadows may lead to the confident assertion that certain pathological conditions exist which have no objective reality.

I purpose taking the subdivisions of the alimentary tract *seriatim*, indicating briefly the information which a physician thoroughly conversant with opaque meal

work should be able to supply.

(1) ŒSOPHAGUS.

Pouches.—The existence of pouches can be demonstrated with clearness, and a negative finding has nearly as much value as a positive.

Obstruction.—The presence or absence of localized obstruction or extensive narrowing can be ascertained. It is often impossible to say whether the condition is spasmodic or organic, or compounded of the two. If any measures, such as the administration of belladonna, cocainizing the gullet, vibration over the spine, etc., cause a complete restitution of the lumen, the diagnosis is clear against organic narrowing; but failure to affect a constriction by these means does not absolutely prove that it is organic.

Irregularity of the lumen extending over some inches, especially if accompanied by the shadow of a mass in the posterior mediastinum, is strong presumptive evidence of carcinoma. But the *absolute* diagnosis of cancer cannot be made by X-rays, which is not surprising, for this also applies to naked eye inspection even after operation exposure.

(2) STOMACH.

Gastroptosis.—The importance of this has been much exaggerated in the past. Care should be taken not to diagnose it from a single examination immediately after the opaque meal. In the case of a nervous patient, I have seen a "dropped stomach" come back to the normal within ten minutes, as the patient's confidence was restored. On the other hand, extreme ptosis, causing delayed emptying and duodenal dilatation, is of moment. It is generally accompanied by sagging of the hepatic flexure and other viscera.

Pyloric Obstruction.—Practically the same remarks apply as were made in discussing the œsophagus, but the diagnosis of organic constriction may often be made with practical certainty.

Ulcer.—An ulcer can be positively diagnosed only if it has a definite cavity, and if this cavity, filled with opaque food, can be seen in profile as an excresence upon the main body of the stomach. A sufficient number of plates, taken at various angles, in a thin subject, should, theoretically, always demonstrate such an ulcer; but, in practice, negative X-ray findings do not rule out even chronic ulcer. A small ulcer cavity, which has escaped observation in profile, may sometimes indicate its presence two to three hours later, when the stomach is more or less empty, owing to a small opaque residue remaining in it. To detect such residues good plates are essential; they are nearly always missed at screen examinations.

Incisura.—This is deep, persistent indentation opposite to an ulcer, and reflexly caused by it. Similar constrictions may be produced by reflex irritation from a diseased appendix or ovary, but in such cases they generally yield to belladonna in large doses. Carman, of the Mayo clinic, holds that an incisura caused by an intra-gastric ulcer is never relaxed by atropine. Whenever X-rays demonstrate more or less persistent spasm in any part of the alimentary tract, the fact is of clinical importance, even though no organic exciting cause can be found for the spasticity. For very often medical measures devoted to counteracting the spasm result in an entire relief of the symptoms, for which the patient is extremely grateful.

Cancer of the stomach, in so far as it can be detected by X-rays, betrays its presence by causing what is known as a "filling defect," *i.e.*, the gastric lumen is encroached on by the growth, and this causes the filling by the barium meal to be imperfect. Certain forms of filling defect are highly characteristic, and unlikely to be caused by syphilis, benign tumour, or adhesions; but, unfortunately, the growth in its earliest stages does not produce any alteration in the gastric shadow. However, by the time there is definite clinical suspicion, there are usually some X-ray signs also.

(3) DUODENUM.

The information to be derived from X-ray examination of the duodenal region, though it often lacks definiteness, is nevertheless of great practical importance.

The first and second portions of the duodenum may sometimes be seen in active contraction, vainly endeavouring to drive a bolus onward, and a subsequent plate may show great dilatation of these parts of the gut. At operation, no adequate mechanical cause may be apparent; nevertheless, a gastro-enterostomy relieves all the symptoms.

The duodenal cap may be shown to be contorted and contracted; but, unless the actual pocket of an ulcer is seen, it is almost impossible to state exactly what pathological condition will be found. A skilled radiologist is, nevertheless, in many cases, able to say that organic changes of a pathological nature have occurred, though whether the mischief is gall-bladder trouble with or without calculi, duodenal ulcer, or simply "idiopathic" dilatation of the duodenum he may be unable to distinguish.

The present writer, when he finds certain X-ray appearances in the duodenum, invariably informs the medical man in charge of the case without waiting for confirmation, his reason being that in two such cases which came under his observation sudden accidents occurred; in one a fatal perforation, in the other a very severe hæmorrhage.

(4) SMALL INTESTINE.

The greater part of the small intestine is usually not definitely outlined by an opaque meal, though some of the coils may be visible for a short time if the stomach empties rapidly, and there is a form of malignant disease known as "leather-bottle cancer" in which the whole of the gastric contents are shot through the rigid pylorus within a few minutes. Apart from this, individual coils may be outlined owing to partial obstruction, either by adhesions or new growth.

The barium food normally becomes visible again in the terminal ileum. Much controversy still rages as to what constitutes iliac stasis. Without in the least denying the possible importance of lesser degrees, the present writer is, at any rate, quite certain that the presence of opaque food in the ileum 20 hours after the stomach is empty is definitely pathological, and always gives rise to symptoms of some sort, very often to those of duodenal irritation suggesting ulcer.

(5) APPENDIX.

If the patient is carefully prepared by castor oil and fasting, it is probable that, provided the appendix is normal, it will from time to time contain barium food, though it may not happen to be seen filled at the times of examination. On the other hand, if its cavity is obliterated by disease, it will not be seen, but negative evidence is in this case only of minor value.

If the appendix is freely movable and deep pressure over it does not cause pain, the presumption is that it is not diseased. If it is tender, and bound down either locally or generally, it is almost certainly the seat of chronic inflammation. This presumption is strengthened if it is found to contain a residue many hours after the cæcum is empty.

There can be no doubt but that a large number of appendicectomies are performed when this organ is not at fault, and the patient is consequently often worse rather than better. If every case, in which an "interval operation" is contemplated, were subjected to opaque meal examination, I feel sure that much unsatisfactory surgery would be avoided. On the other hand, abnormalities in other parts of the alimentary tract would often be discovered; and additional information might be obtained even in cases when the X-ray examination confirmed the clinical by incriminating the appendix.

(6) LARGE BOWEL.

Stasis.—While general stasis in the large bowel may be detected in many ways, localized delay can be diagnosed only by the opaque meal. Stasis in the cæcum is of importance in confirming a diagnosis of appendicitis or perityphlitis; or, in a case of simple atonic dilatation, in showing where to apply massage and electrical stimulation. At the other end of the large gut, the detection of rectal stasis—the dyschezia of Hurst—enables the appropriate therapeutic measures to be used.

The presence of *diverticulitis* may be suspected while the meal is *in situ,* and confirmed after its evacuation by the residual presence of clusters of the characteristic small round masses of barium food.

Narrowing of the lumen and apparent filling defects may be noticed, and lead to a suspicion of organic obstruction and new growths. But such a diagnosis should not be definitely made unless confirmed by opaque enema.

The *opaque enema* is essentially concerned with organic conditions as opposed to functional; and for this reason every effort should be made, previous to its administrations, both to get rid of any fæcal masses and to eliminate spasm. Belladonna to the limits of safety, castor oil by the mouth, and oil enema should all be employed, and that more than once, before one concludes that any obstruction or alteration of contour is organic.

In opaque meal work, neither screen nor plates can be exclusively relied on. To demonstrate small ulcers, photography is essential; on the other hand, palpation under the screen on the couch is necessary to prove normal mobility of organs or limiting adhesions, as well as to show the relationship, if any, between points of tenderness and parts of the alimentary tract. Reverse peristalsis, too, cannot be diagnosed from plates.

It should never be forgotten that the X-ray examination is only *one* of the means to a correct diagnosis. The radiologist must be a competent physician, or his reports will be worth little. But he must be careful to make it clear what is fact and what opinion. For instance, he may assert as a *fact* that a large penetrating ulcer of the lesser curvature is present, for the X-ray signs of such a condition are to all intents pathognomonic; but if he goes on to say this ulcer is malignant or the reverse, this can only be an opinion based on the X-ray findings in conjunction with what he knows of the history and the results of clinical and chemical examination.

The practitioner in charge of the case is bound to accept the statement as to presence of an ulcer; the opinion as to its nature is on an entirely different footing. Nevertheless, the opinion of a man who is a physician before he is an X-ray worker, who takes careful histories of all his cases, and informs himself whenever possible as to subsequent operative results, comes to associate certain symptom-complexes in his mind with definite organic findings, and his opinion is therefore likely to be of value.

In conclusion, it should be noted that a patient is —or should be—sent to the radiologist in order that he may help to elucidate the diagnosis, and not for a specific kind of examination as an end in itself. In many cases it is impossible to be satisfied that all has been done unless the gall-bladder region is examined, and this cannot be done satisfactorily while the opaque meal is *in situ.* A positive diagnosis of gall-stones is of high value, though the converse is not. Often, however, an enlarged gall-bladder may be shown though no calculi are visible. In other cases, kidney and ureter examinations may have to be undertaken in order to clear up the cause of an obscure abdominal pain. The chest should always be looked at on the screen. Recently, I came across a case in which difficulty of swallowing was complained of. Examination by opaque bolus was negative, but an enlarged atonic heart was seen. After a long holiday the patient showed a normal cardiac shadow, and all œsophageal symptoms had disappeared. In another instance, the timely discovery of a thoracic aneurism led to an intended operation being cancelled.

Frequently it happens that an opaque meal examination, or some portion of it, must be repeated in order to clear up doubtful points. It must be remembered that each phase is a fleeting one, and that by the time plates are prepared and studied, the particular set of appearances which they represent has in all probability changed.

The complexity of X-ray investigation of the abdomen increases year by year, and will in all probability continue to do so. This means corresponding increase of expense. But, in the long run, nothing can be so expensive to patients, and to the nation in general, as the performance of unnecessary operations, or failure to carry out those which are essential.

Acute Appendicitis: A Study of 370 Cases.

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THIS paper is based on 370 cases of acute appendicitis upon which I have operated during the eighteen months from June 1, 1917, to November 30, 1918. Only acute cases are considered, and I have attempted to convey in the following classification the stage at which operation was performed. It is, perhaps, not a scientific one, but serves to emphasize the all-important point of early operation.

1. *Acute Inflammations.*—In these the appendix was red, swollen, stiff, and with a little roughening of its peritoneal coat by the inflammatory process. Usually the peritoneum contained more or less turbid fluid, which did not smell. Sometimes the appendix showed a distinct constriction, the distal part being tightly distended and always inflamed. This was the commonest type: 176 cases; no deaths.

2. *Gangrenous Inflammations.*—Here the appendix showed large or small areas of gangrene, but no actual perforation. It was distended as in Group 1, and evidently on the point of rupture. Infection had begun beyond it in the peritoneum, which showed turbid fluid becoming offensive—smelling of a B. coli infection. In this class there was no definite abscess, some reddening and distension of the adjacent coils of small intestine, but no general peritonitis. This was not a common type: 17 cases;

1 death.

3. *Perforative Inflammations.*—There was a definite perforation, varying from a complete sloughing off of the appendix to a minute hole which had obviously only just occurred; in fact, once or twice it occurred during the removal of the appendix. The extravasated matter in the early cases was a little dirty brown offensive fluid in the immediate neighbourhood of the appendix; from this there was a progressive march through localized abscesses to spreading peritonitis, and beyond this to generalized suppurative peritonitis. In the earlier perforations the pelvis usually contained slightly offensive turbid fluid. This group comprises 158 cases; 18 deaths.

Taking these groups together it will be seen that the total mortality was 5 per cent. Earlier diagnosis and prompt operative treatment should bring all the cases into Group 1, in which the mortality was nil in this series. It is not too much to say that the mortality of acute appendicitis ought to be reduced to at most 1 per cent. Delay is responsible for this avoidably large mortality, and the modern text-books and teaching are largely responsible for this delay.

There is no acute abdominal condition which is easier to diagnose than acute appendicitis, and it is certainly one of the commonest. The initial symptoms of acute abdominal pain at first general, and tending to localize in the right iliac fossa, accompanied by vomiting, tenderness, rigidity of right rectus, and temperature, especially in young adults, mean acute appendicitis, often an acutely distended appendix, the further course of which it is beyond the power of anyone to predict with assurance. Sometimes the pain is of a colicky nature, which indicates that the appendix is trying to empty itself against obstruction; later, if the condition is going to become acute, the pain will assume a constant and more severe type. In the earlier stages of this variety the appendix is merely tightly distended and will almost certainly go on to perforation. Anything beyond the stage at which acute inflammation is present should be regarded in the light of a complication, which it is a reproach to a medical attendant to allow to develop. Yet we still read in medical text-books, under symptoms of acute appendicitis, of abscess formation and such like, and, under treatment, of the time to operate and whether to do so at all. It should be possible with better teaching and more accurate observation to bring all the cases into the group where the patient can be told that operative treatment means no risk, whereas delay may mean a very considerable one, and I think there would be few patients who would themselves be responsible for the delay under such circumstances.

PATHOLOGY.

Appendicitis is a combination of obstruction of the lumen of the appendix, and infection behind this. The lumen is, I believe, always more or less obstructed before an acute attack of inflammation occurs; this obstruction is brought about in many ways. In most cases it is the result of former slight and usually repeated infections—not sufficient to call acute—but enough to give rise to some subsequent fibrosis of the appendix wall. The effect is to cause narrowing of the lumen at some point. Or the infection may penetrate to such a depth as to cause a reaction on the part of the peritoneum resulting in an adhesion which eventually contracts and causes a kink, giving rise to narrowing of the lumen. Some of these bands may be congenital, but, I believe, the majority, if not all, are due to infection.

McCarty[1] gives convincing evidence of the inflammatory origin of this partial and complete stenosis in a large series of cases. Rosenow has shown that the wall of the appendix contains in its substance a specific streptococcus, and it is probable that some increased activity of the organism is responsible for the lesions under consideration. Tubercle and carcinoma also are responsible for a few cases of stenosis; two of the former and one of the latter are in this series.

Whatever the nature of the obstruction fæcal material accumulates behind it. There is no doubt that fæcal material enters the appendix; this organ can be filled with barium and shown on an X-ray plate. There is no doubt either that the normal appendix can empty itself entirely and painlessly; but when some stenosis has occurred, as indicated above, fæcal material which squeezes through from the cæcum is retained at least in part, and mixing with the secretions from the appendicular wall eventually becomes inspissated (as fæcal matter elsewhere when retained) and moulded into the well-known concretions; the date-stone shape is given by the cavity in which it lies. It is, no doubt, true that extraneous matter such as fruit seeds, thread worms and the like, are found in these concretions, but the presence of these is not essential to their formation.

When a concretion has formed, the train is laid for an acute attack of appendicitis; the appendix, in attempts to rid itself of its contents, drives the concretion into the narrowed lumen; it becomes impacted, in fact, as a stone in the cystic duct. This gives rise to acute abdominal pain, at first colicky in nature, later becoming constant, but the really serious consequences arise from the effects of retention behind the impaction. Here bacterial activity is much increased, and the rapidity and severity of the attack depends on the virulence of the imprisoned organisms at the time. Wilkie[2] calls attention to this appendicular obstruction, and by experiments on cats, using the bilaterally excluded terminal ileum as an artificial appendix, has arrived at the following conclusions:—

> "In experimental obstruction in an artificial appendix the changes vary greatly according to the nature of the diet of the animal previous to experiment, rich proteid diet being associated with much more rapidly destructive changes than carbohydrates.
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> "Undigested protein, putrefactive bacteria, and an alkaline reaction together produce rapid gangrene in the walls of an obstructed organ. The prevalence of the severer forms of acute appendix in Western as compared with Eastern peoples is probably to be explained by the animal diet indulged in by the former.
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> "On the same lines may be explained the increasing frequency of such diseases in industrial areas and its relative frequency in the male sex."

And, I think, the higher mortality in the male.

Wilkie differentiates between acute appendicitis and acute appendicular obstruction. I believe the former is also obstructive, but not to so decided a degree. Swelling, as occurs in all glandular tissue in acute infections such as fevers of various kinds, may give rise to sufficient obstruction to lock up temporarily bacteria, etc., in the appendix and so produce the so-called catarrhal appendicitis. The popular belief in a chill preceding appendicitis may thus have some foundation in truth, and those cases of acute appendicitis, which occur at the time of menstruation, would be accounted for on this congestive theory, the congestion in this case occurring *viâ* the appendiculo-ovarian ligament of Clado. Injury, too, might act in much the same way. It is stated that during the late influenza epidemic there were several cases of acute appendicitis, which were regarded as complications directly due to the attack of influenza. If a well-marked stenosis already exists in the appendix, it is obvious the obstruction would be more complete, and so the attack be more severe.

The further progress depends on the bacterial activity behind the obstruction; when the obstruction is not severe and the bacterial activity only slightly enhanced, the condition will probably settle down leaving the appendix more or less damaged, and liable to subsequent attacks with increasingly severe consequences.

In Korte's cases (Nordmann) there had been only one preceding attack in 75 per cent. of the cases which ended fatally as the result of recurrent attacks.

The amount of damage to the appendix varies with the intensity of the infection from slight thickening of the wall to partial or, after repeated attacks, complete obliteration. I have several times seen, as the result of former attacks, the terminal part of the appendix more or less severed from the rest, and once or twice I have seen this detachment complete. An appendix completely obstructed in this way may become distended with mucus, the bacteria having died out, with the production of a mucocele. With rather more active bacteria an empyema results which may prove rapidly fatal by perforation, or it may be slow in its progress and become smothered in adhesions. Great activity on the part of the locked up bacteria results in rapid sloughing of the appendix; this great activity is probably dependent partly on the amount of fæcal material in the obstructed lumen.

The reaction of the peritoneum to the inflammatory attack results in adhesions kinking the appendix or completely burying it, and causing adhesions among the coils of small intestine. The omentum not infrequently adheres to the region, and sooner or later acute intestinal obstruction by band or kinking may result from one or other of these.

The tip of the appendix itself may become adherent to the mesentery or elsewhere and act as a band. I think the adhesion binding the terminal ileum to the pelvic brim is frequently the result of appendicitis; this so-called Lane's kink is undoubtedly sometimes congenital, but the frequent association with a damaged appendix, as well as the not infrequent intimate adhesion of the band producing the kink and the appendix, strongly suggests an inflammatory origin in these cases. I have seen altogether some half dozen cases in which this kink has caused acute intestinal obstruction not merely by kinking, but by a rotatory twist of the intestine on its long axis at the same time.

The organisms concerned in the acute inflammatory processes, which supervene in an acutely obstructed appendix, are chiefly streptococci and bacillus coli. It is possible that the streptococci, which, according to Rosenow, have a selective affinity for the walls of the appendix, are those primarily stimulated to unusual activity owing to the disturbance of circulatory equilibrium following on the obstruction; the B. coli, however, soon gain access to the lymphatics of the appendix wall, either through abrasions in the mucous membrane resulting from the distension of the appendix or through spaces opened up from the fretting of the wall by the concretion, and growing apace soon overwhelm any other organisms, and give the odour characteristic of their growth to the fluid which eventually appears outside the appendix in the peritoneum, and also to the abscesses which may form. Prior to the appearance of this B. coli smelling fluid, there is often present a thin, greasy, non-odorous turbid fluid, which is poured out by the defensive mechanisms of the peritoneum, and which, according to Dudgeon and Sargent,[3] contains staphylococcus albus. The protective nature of this fluid is partly displayed in the assistance it gives to the omentum and intestinal coils in the process of gluing themselves together, and so forming a barrier to the spread of the infection.

THE FURTHER COURSE OF THE DISEASE.

If the attack does not settle down, what I prefer to call complications arise; the appendix becomes more and more distended, and the blood supply interfered with, either from the acute distension or thrombosis in the veins, until it perforates. The perforation may be anything from a minute hole, perhaps, into the appendicular mesentery, or into the general peritoneal cavity, to a complete gangrene and sloughing off of the whole organ. The further course depends on the fight the patient's defensive mechanism is able to put up, and the severity of the bacterial invasion. Usually the mischief is localized, the omentum wraps itself round the appendix sealing up the hole, or isolating any abscess that may form; the intestines may also come to the rescue, gluing themselves together, and a large or small abscess results in the pelvis, behind or to the outer side of the cæcum, or, in a less frequent but more serious spot to the inner side of the cæcum; or there is a combination of these.

In the series of cases under discussion 89 developed abscesses—58 of which were in the pelvis; 10 of these had other abscesses as well—8 of which were in the right loin, and 2 at the root of the mesentery.

The other 31 of the 89 cases of abscess were purely other than pelvic; the majority were behind or to the outer side of the cæcum, and varied in size from an ounce or two of pus just above the pelvic brim, or lying in the right kidney pouch, to an abscess filling the whole of the loin as far as the under surface of the liver.

I should like to emphasize this not infrequent association of two or more abscesses. I have on several occasions seen a pelvic abscess when the appendix has been lying with its tip in relation to the kidney, and even though there has been no abscess in the latter situation. I am not sure of the significance of this, but I think it is probably due to the protective fluid which is poured out freely in the early stages of an acute appendicitis. This gravitates to the pelvis. Probably many of these collections are absorbed, but occasionally they become infected from the limiting intestinal coils, and require drainage.

Abscesses vary in size; those in the pelvis and beneath the liver are apt to be the largest, *e.g.*, from the pelvis an abscess may rise into the abdomen reaching to the umbilicus, and simulating very closely a dilated bladder; at the same time it pushes the bladder, rectum, and uterus down, causing tenesmus, diarrhœa, frequent and painful micturition, or even retention.

Abscesses, if not treated surgically, may burst on the surface of the body in various positions, *e.g.*, on the anterior abdominal wall, in the loin, or they may track through the pelvic fascia and discharge on the buttock, or in the ischio-rectal fossa; or, again, they may burst into the rectum, cæcum, vagina, bladder, or the small intestine. I have operated on a case which showed quite clearly the fistulous track between the appendix and the terminal ileum. An abscess may rupture its limiting adhesions, and so give rise to general peritonitis. Other consequences of leaving an abscess are pyelophlebitis, septicæmia, pyæmia, and death.

Left-sided abscesses are uncommon apart from a general peritoneal infection, but the possibility should be kept in mind, for a long appendix may lie across the brim of the pelvis, and, becoming inflamed, cause an abscess in the left iliac fossa; there are, too, the rare cases in which the cæcum lies on the left side. There was one case of the latter condition in this series, in which none of the large intestine was to the right of the middle line. The cæcum was in the left iliac fossa, the ascending colon ran vertically up to the splenic flexure, and down again, as though there were no transverse colon at all. The appendix was perforated, and there was general peritonitis, the intestines being red, distended, and here and there showing patches of lymph gluing coils together; there was no collection of pus, and the peritonitis did not seem to be very severe, the wound was, therefore, closed after removing the appendix. In one case of this series an abscess was found outside the peritoneum, between it and the pelvic fascia. It is not uncommon to find enlarged glands in this situation, and I think this case was probably one in which such a gland had suppurated.

In three cases an abscess formed in a hernial sac on the right side; in one the inflamed appendix was in the sac, in the other two pus had spread from an intra-abdominal appendicitis.

Subphrenic abscess and empyema occasionally occur.

Secondary hæmorrhage from the deep epigastric vessels is anot er results of sepsis, which was met with once in this series.

A further complication of an attack of acute appendicitis, which does not settle down, is general peritonitis. This may be either generalizing or generalized; it is not always easy to say at operation, through the limited incision used, which is present. The former, with correct treatment, may be cut short before it becomes generalized, and statistics, which refer to these two conditions separately, are certain to be inaccurate. In this series the two are considered together; there were 33. They all presented signs and symptoms pointing to infection which had spread well beyond the primary focus: flushed face, rapid pulse, increased frequency of respirations, raised temperature, frequent vomiting, distension, constipation, rigidity and tenderness of the abdominal wall extending to the left of the middle line, but not always to the whole abdominal surface; in the more desperate cases the patients had the sunken eyes, apathy, clammy skin, subnormal temperature, weak, thready pulse, etc. of advanced toxæmia. Some of these cases had clearly generalized suppurative peritonitis on admission, others were as clearly not in this advanced stage. There were 8 deaths amongst these 33 cases—24 per cent. This is not the hopeless position that it used to be, which is due to the present day treatment to be detailed later.

With regard to these cases, they are more common in children. Collinson[4] has pointed out that rigidity particularly is a sign which in children often misleads; frequently it is scarcely present at all, leading one to underestimate the severity of the attack. There is here, then, a dangerous possibility of delay, and the development of the child's omentum increases the risk of this delay, for it is comparatively small in them, and I have noticed a tendency it has to recede from the infected area; in adults it is very seldom that the omentum is not on the spot. This all makes it imperative to operate on children at the earliest possible moment after an acute appendicitis has begun.

There is one complication of which I wish to speak, and that is acute intestinal obstruction. This is not common but is very serious, being associated with a high mortality.

There were six cases in this series—in two obstruction was present at the time of operation. In one of these two the inflamed appendix was adherent to the mesentery by its tip, acting as a band across the lower ileum; in the other the condition was similar to what Sampson Handley[5] has called ileus duplex. Both cases recovered; one after appendicectomy and the other after freeing the coils of intestine and appendicectomy.

The other four were post-operative, there were three deaths, and one recovery. These four were all cases of perforative appendicitis in which the appendix was removed at operation, and the pelvis drained. In all adhesions were the cause of the obstruction, usually in the form of a complex matting of coils; in one there was a well-defined adhesion, which had not only tied down the ileum in the neighbourhood of the pelvic brim, but had rotated it on its long axis as well. This is a very common method of production of obstruction, especially in tuberculous peritonitis cases. These post-operative obstructions are definitely mechanical, and not merely paralytic.

There is no doubt delay is the cause of the high operative mortality in these cases. The onset is rather insidious; it is clear something is wrong with the patient; possibly only a little colicky pain and distension, and the eyes appear somewhat sunken, still the bowels act pretty well, and there may be no vomiting. The first vomit may be fæcal. It is in this more or less latent period that operation should be performed; these patients go downhill with great rapidity when the nature of the lesion is obvious, and operation at this late stage is accompanied by a mortality of something like 80 per cent.

The obstruction usually develops within the first month after the appendix operation, often within the first week. It may be enough merely to free the adhesions, but if the patient is able to stand a longer operation, lateral anastomosis to short-circuit the involved part gives a better prospect of cure.

There is one point which I wish to emphasize, viz., patients are put into a much better condition for operation after two or three hours' continuous saline infusion given both subcutaneously and rectally. The amount of fluid should be pushed, so that they get as much as two or three pints in an hour. I am quite convinced that this is a point of the highest practical importance. Another is washing out the stomach, and repeating it if necessary; this is essential if an anæsthetic is to be administered.

The treatment adopted in the four cases referred to above was merely freeing the adhesions in two cases—both died; they were too ill to stand more. The third case had a lateral anastomosis made between the ileum and transverse colon, but died. The fourth recovered after an ileo-colostomy had been performed. This was a case late in the series, and saline infusion was pushed energetically before operation.

Before discussing the treatment adopted in these 370 cases, let me mention one or two points of interest.

I have drawn up a table showing the sex and age of all the cases as follows:—

	Male.	Female.
0 to 5	2	3
5 to 10	31	16
10 to 20	84	67
20 to 30	25	46
30 to 40	24	29
40 to 50	19	12
50 to 60	9	5
60 to 70	2	1
Total	196	179

The youngest in the series was a male of 6 months;

the oldest a male of 66 years.

It is interesting to note the greater incidence amongst females between 20 and 30 or so. I think it is probably connected with active menstrual life, because it is a common occurrence for an acute attack of appendicitis to begin about the time of the monthly periods.

As regards the Mortality.—There were 19 deaths: 13 males—6·6 per cent.; 6 females—3·4 per cent., *i.e.*, 5 per cent. for all cases.

Of the 19 deaths, none occurred in Group 1 (*see* classification); 1 in Group 2; 18 in Group 3.

The death in Group 2 was in a case of gangrenous but not perforated appendix, lying along the outer side of the cæcum, with its tip beneath the liver. The appendix was removed and the wound stitched up. The patient subsequently developed a large pelvic abscess, which was drained, but the patient succumbed. The site from which the appendix was removed, remained free from abscess formation. This is an instance of the condition to which I have referred in an earlier part of this paper.

The 18 deaths in Group 3 were due to acute obstruction in three cases, empyema in three, secondary intra-abdominal abscesses in three, and the rest were due to general suppurative peritonitis, usually accompanied by broncho-pneumonia.

The position of the Appendix was noted in 161 cases. In 71 it hung down into the pelvis, or lay along the brim; in 80 it was either behind or to the outer side of the cæcum; and in 10 it lay internal to the cæcum, either in front of or behind the terminal ileum.

The conditions with which the diagnosis of acute appendicitis was confused, were mostly cases of acute salpingitis. This does not usually call for operation, but no serious consequences result if an operation is needlessly performed. Frequently the appendix was found reddened by contact in these cases, and

I have seen it stated that an acute attack of appendicitis may result from this; I have never come across this, and I am very sceptical about its occurrence.

Acute pneumonia is another trap for the unwary, more particularly in children, and it is a serious mistake to administer an anæsthetic in these cases; but it should be sufficient to bear in mind the possibility of such an error. However, mistakes will occasionally continue to be made in this respect, for an appendix lying well up beneath the liver is apt to cause secondary inflammatory manifestations in the chest, making a correct diagnosis in the early stages a very difficult matter.

Perforated duodenal and gastric ulcers, some cases of diverticulitis, acute cholecystitis, and perforated neoplasms of the large intestine have all been mistaken—these are, however, all conditions requiring surgical aid.

Another condition, which may be confusing, is acute pyelitis. These cases may strongly suggest a high lying appendix; an examination of the urine will easily reveal the true state of affairs.

TREATMENT.

All the 370 cases were submitted to immediate operation. 227 were treated by appendicectomy and closure of the wound. 3 died—a gangrenous case reported above and 2 from general peritonitis. 124 were treated by appendicectomy and drainage. 15 died: 3 from acute obstruction; 1 from empyema; 3 from secondary abscesses; 8 from general peritonitis. 19 were merely drained, the appendix not being sought for, either because the patient was too ill or the adhesions too firm. 1 died from general peritonitis.

Of the perforated cases, 38 were treated by appendicectomy and closure of the wound—with two deaths; these could probably have been saved by drainage. On the other hand, of the remaining perforated cases, there were probably several which could have been closed with safety. This is largely a question of judgement and experience, and on the whole the number of cases drained was less numerous in the latter part of the series, though I still prefer in doubtful cases to drain for a few hours.

The wounds of the Group 1 type are always closed; the kind of case amongst the perforated, which, again, I have no hesitation in closing, is the one that has apparently recently ruptured, and where there is only a small quantity of pus or a little dirty brown fluid in the immediate neighbourhood of the appendix; there may be a considerable quantity of turbid, non-smelling fluid in the vicinity and in the pelvis, which should be mopped out with gauze. It is not common for an abscess to form in the cases after operation. What does happen not infrequently is that the cellular tissue of the abdominal wall becomes infected, the wound looking red and swollen four or five days after operation; on removing a stitch or partly opening the wound a varying quantity of foul smelling pus comes away. With 10 per cent. saline fomentations this clears up in about a week.

If a tube had been used from the first, the wound would have been rather longer in healing, and there would have been a greater risk of intestinal obstruction (not a great one, perhaps), probably owing to denser adhesions forming, and a distinctly greater risk of ventral hernia. Scudder and Goodall[6] give the following figures:—

Of 640 cases 10 per cent. developed hernia through the scar.
Of the 309 drained cases hernia formed in 17 per cent.
Of the 331 undrained cases hernia formed in 3 per cent.

When there is a definite abscess I always drain, and if there is a definite general peritonitis I leave in a pelvic tube as well. When the peritonitis is clearly only spreading, I usually close the wound.

The doubtful cases are those in which there is a fair amount of pus without any definite limitations, especially when it shows a tendency to collect in the pelvis, and those in which there is a considerable quantity of turbid, somewhat offensive fluid, which is collecting in the pelvis, and showing a tendency to become loculated by adhering coils of small intestine. I have closed such cases, but have had to drain some of them later, and I think it is better to drain at first for a few hours.

With regard to the technique of operation. I almost always use the vertical incision, displacing the rectus inwards. Having opened the peritoneum, I clip the edges to a tetra cloth, or better, to a mackintosh cloth (rubber covered with gauze), in such a way as to hide completely the subcutaneous and muscular layers. I have tried other methods, such as rubbing in sterile vaseline and washing it out afterwards with 10 per cent. saline, rubbing in iodoform and glycerine, etc.; but nothing is so efficacious as this mackintosh barrier, though the vaseline method might be used as an additional safeguard.

Having so arranged things that any infective material is discharged without touching any part of the wound, I next feel round to get some idea as to whether there is likely to be pus present or no. If there is, gauze packing is introduced in sufficient quantity to form a thick barrier to prevent the pus, as it wells up, from contaminating the adjacent coils of small intestine, or the general peritoneal cavity beyond. Any freed pus is carefully mopped up and the appendix sought for. This may be a very difficult stage of the operation, but provided the patient is sufficiently fit, and the adhesions not excessively firm, the appendix should always be found and removed. I have several times opened an abscess and discovered another quite distinct one around the appendix, which would certainly have been left had the appendix not been unearthed.

The best and surest way of finding the appendix is to hook up the cæcum with a finger; it is easy to do this, if the finger is made to catch against a muscular band. With the cæcum fixed it is only a matter of patience and daintiness to free the appendix; its root can always be found by tracing the muscular bands to the apex of the cæcum. Sometimes the terminal part will be found to have sloughed off; it is not a matter of any importance, for it will come away in the discharge, but the root of the appendix must always be secured, otherwise the patient may have other attacks. I have more than once removed practically the whole appendix, or varying lengths of it down to ½ inch, from patients who were supposed to have had the whole organ removed at a previous operation.

Having freed the appendix, the next thing is to divide its mesentery. This is best done by holding it up and transfixing it with a clip, drawing a ligature through, and then passing the clip back through the same hole. After tying the ligature the mesentery is cut distally to it, and the same performance repeated until the cæcum is reached. This method of dividing the mesentery after ligature is very good, and I am indebted to Sir Berkeley Moynihan for it. There is no fear of losing the bleeding point should the ligature break whilst tying it, and this applies with particular force to fat subjects.

If omentum adheres to the appendix, this is ligatured at a short distance away and removed at the same time.

The appendix is now crushed at its base with Doyen's instrument, which leaves a wide groove only crossed by peritoneum, a ligature is tied at the proximal side, and a clip applied to the distal. The appendix is removed by cutting between. The crushed stump should always be tied, otherwise hæmorrhage may occur into the bowel. It should then be buried, partly to prevent adhesions, and partly to prevent a fæcal fistula; the burial is carried out by passing a Z suture of fine catgut taking the sero-muscular coat, and invaginating the stump whilst the stitch is tightened.

This is buried by a second suture of a similar kind. The stump of the cut meso-appendix should be stitched to the cæcum in such a way as to hide its raw face.

The question now arises as to closure of the wound; this is decided on the lines mentioned above.

Particular attention should be paid in this respect to the right flank and pelvis; sometimes it is advisable to drain from the loin by inserting a tube through a separate incision at the posterior part of the lateral aspect of the trunk. If the pelvis is drained, the tube is brought out through the lower end of the incision. The wound is closed by a continuous catgut suture for the peritoneum, one or two silkworm gut sutures picking up all the abdominal wall down to and including the rectus sheath, interrupted catgut stitches for the anterior rectus sheath, and finally Michel clips to obtain accurate apposition of the skin edges.

In the stitched up cases a dry dressing, fixed in place with a glue solution is used; in drained cases, a 10 per cent. saline fomentation is applied, and changed frequently.

All patients are sat up on their return to bed in the Fowler position; the more upright they are placed and the sooner this position is adopted, the better and more efficacious it is.

Most perforated cases are put on continuous subcutaneous or rectal infusion of normal saline; in bad cases both are used for the first 24 hours, whilst nothing is given by mouth so as to keep the intestine quiet. This is carrying out much the same ideas after operation which Ochsner recommends before.

The bowels are encouraged to act by enemata towards the end of the first 24 hours; pituitary extract is also of considerable use at this stage.

General peritonitis cases are treated on much the same lines, but I always employ a pelvic drain, and as much fluid both subcutaneously and rectally as can be hurried in; the Fowler position is essential. I am quite convinced of the efficacy of giving large quantities of fluid in this way.

Secondary abscesses are not uncommon, and will require opening at varying times after the primary operation; a common spot for these to form is the left iliac fossa.

As regards the treatment of abscess cases, ordinarily this is on the same lines as the operation just described; the abdomen, however, requires opening with more than usual care, for it is not uncommon to find the cæcum, or a coil of small intestine glued to the abdominal wall. Generally, abscesses should be opened over the most prominent point. Much œdema of the parietes may be seen, and is an indication that the incision is in the right place. The difference in treatment arises mainly in those cases in which the patient is too ill to stand more than the minimum of interference, or when the abscess is very large, and the walls very firm. In such cases it is better merely to drain, leaving the removal of the appendix to a later date, when sepsis has been eliminated.

In draining these large cavities two tubes are often necessary, and as far as possible they should be placed at opposite ends of the cavity to allow of gentle irrigation during convalescence. Usually it will mean putting one tube into the pelvis and one in the loin. When the walls of the abscess are well defined, healing may be expedited by putting into the cavity one or two Carrel's tubes, and flushing every hour or so with an ounce or two of Dakin's solution. This should only be done in cases in which the abscesses are well shut off.

Large pelvic collections may be drained by the rectum or vagina. I do not often do this, for I have seen rather severe hæmorrhage follow; moreover, it is only the pelvis which will drain, and, as has been pointed out, pelvic abscesses are not infrequently accompanied by abscesses in other parts. The general management of the after-treatment is as stated above.

One of the results of draining an abscess is a fæcal fistula. This fistula may be from the open stump of the appendix or from a damaged piece of the cæcum, or small intestine. The tube is undoubtedly the determining cause in most cases. Probably at operation the intestinal wall has been slightly damaged, and the tube, subsequently pressing against this part, frets through it, or it aggravates the inflammatory process already present to such an extent that the wall softens, and ultimately gives way.

These fistulæ usually heal without any operative treatment; occasionally, owing to the wound in the gut being on the surface of the abdominal wall, healing does not take place; this is especially likely to happen when the small intestine is involved, and the wound in the parietes has gaped, as it is so likely to do under these conditions.

If, after allowing reasonable time for natural healing, and after applying drags to draw the edges of the wound together, the fistula does not then close, it should be packed with gauze, the track excised with an ellipse of skin, and the involved piece of intestine found. Should this be cæcum, it is enough to invaginate the aperture; in dealing with the small intestine either the involved piece should be removed, and a lateral or end to end union done, or the damaged part should be excluded by a short-circuiting operation, with removal of the isolated loop later if the patient finds the small amount of discharge from the fistula too irksome to put up with.

Those cases which recover from operation are usually completely cured. A few return complaining of indefinite pain, which is probably due to incompletely absorbed adhesions.

There are, however, a certain small number who complain of dyspeptic symptoms, which they had before the acute attack.

There was only one in this series that I know of.

The symptoms complained of are "bilious attacks," as the patients usually say, and consist of pain coming on one to two hours after food, sometimes before food, and irrespective of the character of what is eaten. There are no periods of freedom from pain lasting for months, as in duodenal and gastric ulcers; there may be an interval of a few days, but that is all. The pain is felt at times in the epigastrium and at other times in the right iliac region, but it does not keep the patient awake at night. Some part of the symptoms is clearly due to the appendix, but as the removal of this organ does not always completely clear up the patient's troubles, it is obvious that some other organ must also be partly responsible. The very red state of the pyloric half of the stomach, the unusually active peristalsis that one can quite easily see, and the constant presence of a large, fleshy (probably septic) gland opposite the pyloric end of the stomach, and situated about ½ inch from the greater curvature, coupled with the gastric character of the symptoms and the epigastric tenderness, all these seem to me to suggest strongly that the stomach is responsible for the continuance of symptoms in the type of case mentioned above. The probability is that the appendix is the primary offender, and that as a result of pyloric spasm induced by this, as suggested by Moynihan, the gastric mucous membrane becomes hyperæmic (hence the hæmatemesis), and the seat of superficial erosions; the persistence is the result of a wrong diet. Clearly, if the stomach is at fault, these cases should have a more or less prolonged fast, but the tendency of the patient recovering from an attack of appendicitis is to eat heartily.

I suggest that patients who present this kind of history, that is to say, of prolonged appendix dyspepsia, previous to the acute attack or attacks, should have merely water by mouth for several weeks after appendicectomy, provided that the physical state is sufficiently good to permit of this rather stringent treatment.

The ordinary medical treatment of gastric ulcer should be followed.

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Manganese in Suppuration.

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IT is only comparatively recently that manganese in the form of a colloidal suspension, has been introduced into the body. Its value in gonorrhœa, in acne, and in furunculosis, has been demonstrated by McDonagh, and in anæmia by Turner.[1] In THE PRACTITIONER[2] the former also formulated a hypothesis to explain its bactericidal action.

Manganese, which is chemically closely allied to iron, shares with it and several other metals, such as tin, copper, arsenic, etc., the properties (*a*) of combining with oxygen in two proportions, (*b*) of influencing the destruction of micro-organisms in the body. It is suggested that these two properties are related. *In vitro*, manganese is not bactericidal;[3] the action is a "vital" one. The manganese is supposed to act as an oxydizing katalyst or oxidase.

Normally, iron fulfils this function. It is not the combined iron of the hæmoglobin to which reference is had. This being confined to the red muscles, liver, and blood corpuscles, the majority of the cells of the body do not come into relationship with it. The iron of the lymph and plasma, however, comes into relationship with nearly all the cells in the body, native and parasitic; and it is on the oxidase action of this that the bactericidal action must depend. The quantity is very small,[4] and attempts to increase it by injection of colloidal iron are of transitory effect.[5] One may surmise that the physiological mechanism for the utilization and excretion of iron never allows more than the normal minute quantity to circulate. In manganese, however, we have a metal to which the tissues are not accustomed. This metal remains in the circulation for some days after injection. It is supposed to circulate as a hydroxide, adsorbed to a molecule or aggregate of molecules of the protein of the lymph.

An additional property of manganese is its power of stimulating the hæmatopoietic function. This, and the slight leucocytosis which any colloidal injection will produce are doubtless of importance in its action. It appears further, perhaps on account of the stereochemical configuration of the molecule, to have special influence in conditions of coccal infection. Like all the metallic colloids, it is a suspensoid, and will not act as an antigen.

In the series of cases, some of which are detailed below, a technique based on McDonagh's[2] has been followed. The suspension is prepared and isotonized at the time of injection, by mixing the two sterile stock solutions in the syringe. The injection is then made slowly into the gluteus, or, if the patient is confined to bed, into the deltoid. It is painless; sometimes a slight stiffness is noticed for 24 hours. Injections into subcutaneous tissues, intermuscular septa or the periosseous alveolar tissue are attended with pain. I have not given the drug intravenously. The dose has ranged from 0·5 cc. to 5·0 cc. of the one per mille suspension, which dose daily seems to be perfectly tolerated. In chronic cases, several days separate successive doses; from four to seven injections usually suffice. No general reaction has been observed. Post-mortem examination (in a case of double pyonephrosis) showed that the drug is completely absorbed within six days, leaving no trace.

MANGANESE IN SUPPURATION

The metal is stated to be excreted by the kidney; it is probably also largely excreted by the colon.

CASE 1.—A man of 42 with a mild chronic infection of the antra; cacosmia and headache; both antra were opened. Eight days later temperature was 100° every evening, and muco-pus drained freely. Four daily injections of 1 cc. were given. On the thirteenth day temperature rose only to 99°; the discharge was much less. On the sixteenth day temperature was normal, nasal washing returned clear, all symptoms were completely relieved, and the patient discharged well satisfied.

CASE 2.—A patient with infection of left antrum, which improved after drainage and nine months later relapsed. Deafness and a constant discharge of thick muco-pus were principal symptoms. Three injections of ·5 cc. were given on alternate days, then four of 1 cc. every fourth day. The discharge ceased with the sixth injection, and has not returned four months later. The deafness is improving. Protracted and energetic local treatment had not benefited this patient.

CASE 3.—A patient with infections of both antra, which were opened. Three weeks later the condition had not improved. Three injections of 1 cc. were given. The patient felt very depressed after the third dose. She returned home a week later feeling better, but still having a free discharge. One more injection was given. The discharge cleared up, and, although it returns occasionally, she feels and looks very much better.

CASE 4.—Constant rhinorrhœa for 11 years, with headache and anosmia. Three courses of vaccines did not improve the condition, but improvement followed free opening of the accessory sinuses. Eighteen months later the general health was better, but there was still a constant muco-purulent discharge. Two courses of 1 cc. weekly for three doses were given at eight weeks' interval. The discharge cleared up completely, the headaches which had troubled him disappeared, and the sense of smell returned.

CASE 5.—Constant rhinorrhœa and cacosmia of several years' standing, with failing eyesight and memory. The accessory sinuses had been freely opened 10 years ago and entry was free, but they were full of pus; lavage improved but did not terminate the condition. He was ordered three injections of ·5 cc. and six of 1 cc., one a week. He felt very depressed after the injections; no noticeable improvement followed.

CASE 6.—A woman of 23, with history of nasal trouble from childhood. The accessory sinuses were drained seven years ago, but, after long freedom, trouble returned in the wake of an influenzal attack last spring. The patient was very depressed and got constant discharge from the nose, with cacosmia. Lavage improved but did not completely relieve. She was noticeably anæmic—R.B.C., 3,700,000; W.B.C., 8,000. Four injections of 1 cc. on alternate days were given. At the end of the course she felt much better, was obviously less pallid, and had considerably less discharge—R.B.C., 5,100,000; W.B.C., 12,500. One month later the patient was very much better. All lassitude and mental confusion were gone; the colour was good, the discharge nil.

Case 7.—A woman of 23, in very poor condition; the throat was very septic and the uvula gangrenous. Temperature, 101°. The bacteriology was not recorded. Four injections of 1 cc. were given. The gangrenous process was abruptly arrested, rapid recovery followed, and the patient returned home.

Case 8.—A woman of 28 had Ludwig's angina following dental caries, with abscess of the tonsillar gland. Temperature, 103°; pulse, 112. Two ozs. of pus were drained from the abscess. On the fourth day temperature was 97°, pulse 96; the face putty-coloured and lips bloodless, and a free sanguineo-purulent discharge was draining; the woman was very weak and ill. Blood count—R.B.C., 3,800,000; W.B.C., 12,000; Hb, 80 per cent. Injections of 1 cc. for four days were given. On the tenth day she was noticeably a better colour; the discharge was slight in amount, and she felt better; R.B.C., 5,200,000; W.B.C., 14,000; Hb, 80 per cent. She was discharged a week later.

Case 9.—Female, 38. Admitted complaining of swelling on left side of neck. This was found to be an abscess, probably starting from an infected gland in front of the sternomastoid. It was drained. The maxillary antra were both full of pus, and they also were freely opened. Staphylococcus longus was cultivated from all three collections. Nine days later the sinus in neck and the antra were discharging very little; temperature was normal. A week later still the sinus and antral discharge persisted; the temperature showed a constant rise to about 100 in the evening. The skin over the sternomastoid became red and tender, suggesting a spread of infection. The condition got gradually a little worse till 2 cc. of manganese were given intramuscularly every other day. After the first dose the temperature rose to 101°; after the third it came down to normal and remained down; the sinus, 2½ inches deep four days before, was barely discoverable; the antral discharge ceased completely. The patient felt better than she had done for years, and was undoubtedly brighter and a better colour.

The conclusions one was able to draw from the series were:—(*a*) Manganese powerfully increases the resistance of the tissues to antral as to any other local infections. It is, therefore, a valuable adjunct to the surgical treatment of suppuration. (*b*) It further improves the anæmia commonly associated.

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Further Research in the Treatment of Hypertension.

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HYPERTENSION, whether of a pre-sclerotic or sclerotic type, is gradually but surely taking a more and more important place amongst those conditions which cause a large percentage of deaths after middle age, and not an inconsiderable number in earlier years. Before the invention of the sphygmomanometer it was practically impossible to diagnose early cases of the sclerotic type, or to differentiate between these and cases of the pre-sclerotic type, because the errors of the tactile reading of blood-pressure are very great, even when it is practised by those who have special knowledge and opportunity to perfect themselves in this method. Faught[1] says that "even the most experienced have been unconsciously led into grave errors by depending upon tactile sensation, when the sphygmomanometer could have been employed," and L. Krehl[2] says "distinction between internal tension and arterial thickening may elude even the most experienced."

After the introduction of the sphygmomanometer as a clinical instrument, the necessity of blood-pressure readings with reference to accurate and correct diagnosis, prognosis, and treatment, became evident, and these observations began to take an increasingly important position in the science of medicine. At the present time, it is almost impossible to exaggerate the advance in our knowledge that this method of investigation has achieved.

The profession in America take a very grave view of the rapidly increasing number of deaths due to hypertension and arterio-sclerosis. Dr. E. A. Shiply[3] found that, in 1912, one-fifth of all deaths in America were due to diseases of the cardio-vascular-renal type, and he pointed out that from this type of disease the death-rate of persons aged 41 years or more had increased by from 100 to 300 per cent. since 1868. The late Professor Janaway[4] found that the increase in the death-rate from diseases of the arteries in New York City in the decade 1900–10 had increased 396·2 per cent. Statistics such as these prove beyond dispute the highly important part that hypertension and arteriosclerosis take in the death-roll of those who approach, or have passed, middle age.

Despite these facts, it is still not uncommon to find medical men who do not make a practice of taking the blood-pressure records of their patients, and it is still the custom of most, if not all, our insurance offices, to accept lives without previously obtaining a record of their blood-pressure.

One of the main reasons why many doctors take so little notice of blood-pressure records as a clinical guide is, I believe, due to the fact that they are aware that the administration of drugs is of little if any use in the treatment of cases of arterio-sclerosis. They have been educated in the belief that the changes in the vessel-walls, which produce this disease, are a necessary accompaniment of increasing years, and must be looked upon as an evil which cannot be dealt with, even in cases in which they set in many years earlier than is common. Those who take this view lose sight of the fact that, in every case of arterio-sclerosis, there is a pre-sclerotic stage of hypertension, which, as Clifford Allbut[5] says, "if caught in time is, if not always, yet usually curable." These cases can be cured by the administration of suitable drugs, a regulation of the diet, and adherence to healthy rules of living, and each case so treated means a gain of some years in the life of the patient and, if not always an absolute cure, yet a very material retardation of the sclerotic process. The only sure method of not overlooking these cases is the routine use of the sphygmomanometer.

When once arterial degeneration has begun, no treatment will cure it, and the best result we can expect from suitable treatment is to retard the development of the disease and relieve the patient of those trying and alarming symptoms which are caused by hypertension. This can only be done by methods which lower the blood-pressure and, at the same time, strengthen the cardiac action, thus relieving heart-strain, improving the circulation, reducing venous congestion, and accelerating the oxygenation of the blood, and excretion of those toxic materials which set up high blood-pressure and spasm of the vessels.

Huchard[6] lays great stress upon massage and baths for combating hypertension, and I myself have found no treatment which gives a better or more lasting result than a course of "Nauheim" baths by itself, or combined with massage and resistance exercises By these methods the action of the heart is slowed and strengthened, and the blood-pressure is definitely reduced. The effect lasts for a longer time after each succeeding bath, and the blood-pressure, although rising again somewhat after the bath, tends to become lower and lower as the course proceeds, so that at the end of five weeks' treatment it is usually materially reduced. This satisfactory condition will remain for a long period, sometimes even for several years after the course is finished, provided that the condition of degeneration in the vessels is not very advanced, and the patient follows the directions of healthy living, which help materially to keep down hypertension. The result of treatment lasts longer in the young, middle-aged, and those whose vessels are moderately healthy than in the old and decidedly sclerotic cases. Continued indulgence in over-feeding, sloth, or alcoholic excess, will hasten the return of symptoms such as vertigo, headache, loss of memory, insomnia, dyspnœa, dyspepsia, palpitation, or angina; these being some of the commonest conditions caused by arteriosclerosis or hypertension.

A large majority of the victims of this disease are found amongst middle-aged people who have made a constant practice of eating too much and taking little or no exercise, these conditions being as prone to set up arterial degeneration as alcoholic excess. Mental worry and anxiety are also common factors in its development, as also is syphilis.

Some cardiologists have maintained that "Nauheim" baths, more especially the effervescing variety, raise the blood-pressure in cases of hypertension, instead of lowering it. The object of this paper is to prove that this assertion is *absolutely wrong*, and that those who have made it cannot have had a practical knowledge of the treatment, but must have founded their belief on hearsay or incorrect data accepted with insufficient proof of their reliability.

I have made a careful study of the effect of every variety and strength of "Nauheim" bath, both upon the maximum and minimum blood-pressures, in a very large number of cases under my care, and *I have never found a single instance in which both pressures did not fall in the bath,* sometimes to the extent of 30 to 50 mm. Hg., and very much more in exceptional cases (Case 3). I have made all my observations with an Oliver's mercurial sphygmomanometer and Oliver's phonendoscope.

The routine procedure has been to take the rate of the pulse and the blood-pressure on the left arm of the patient whilst he is lying in bed before the bath, then to take them on the same arm at intervals of about two minutes whilst he is lying in a similar position in the bath.

I have not found any record of observations made both before the bath as well as *actually whilst the patient is in the bath,* and it is only by taking records in this way that the true effect of the baths upon hypertension, both sclerotic and pre-sclerotic, can be ascertained correctly. I maintain that all deductions based upon a less careful and thorough investigation are utterly misleading, and it is because of the immense importance of a true knowledge of the effect of "Nauheim" baths upon hypertension, and the wide range of usefulness of this treatment in combating a disease, in which drugs have proved useless except in its earliest stage, that I lay such stress upon this all-important point.

In most of the modern books by English authors upon diseases of the heart and vessels, the "Nauheim" treatment is either ignored or receives very faint and cursory praise, and in some it is actually stated to be of no special value. I am convinced that opinions of this kind are the result either of a definite lack of practical experience, or of an attitude of mind which regards accurate and minute scientific diagnosis as of much greater importance than treatment.

There is no doubt that the importance of accurate and scientific diagnosis is very great indeed, and I find that the employment of the latest methods of investigating cardiac and vascular diseases are all-important as aids to proper treatment, but they cannot be of much benefit to patients in the hands of those who regard them, not as means to an end, but as the end itself.

The "Nauheim" baths are not only useful as a highly successful method of treating hypertension, but their effect upon hypertension enables one to come to a definite conclusion whether a case is of the pre-sclerotic or the sclerotic type. In cases of the pre-sclerotic type, in which the raised tension is entirely due to spasm of the vessels, and the vessel walls are healthy, the blood-pressures, both maximum and minimum, will drop to normal during a bath, however high they were before it (Cases 2 and 3); if, however, the hypertension is due partly to spasm and partly to arterio-sclerotic changes, the vessels, not being as elastic or expansile as in health, do not respond to so great an extent to the effect of the bath, and consequently the blood-pressure in such cases never falls to normal in a bath. Cases 1 and 4 are typical of arterio-sclerotic hypertension, and it will be observed that in these the maximum blood-pressure does not fall to normal during the bath (Tables I., VI., VII., and VIII.). Cases 2 and 3 are of the pre-sclerotic type, Case 2 being one of pre-sclerotic hypertension and dilated heart, and Case 3 one of a very fatty dilated heart with great hypertension but no arterio-sclerosis. Although the pressure in these cases was abnormally high before the bath, it dropped absolutely to normal, both in the maximum and minimum readings, during the bath. I had never before seen a case in which the pressure fell to so great an extent in the bath as did that of Case 3, and I was surprised to note that the patient did not experience the slightest discomfort, although the fall was so rapid. Cases showing so decided an alteration in the blood-pressure in so short a time are very rare. I have used the terms *maximum* and *minimum* blood-pressure in place of *systolic* and *diastolic,* because I do not believe that these two readings have any relationship to the systole and diastole of the heart.[7] The minimum blood-pressure is indicated by the reading on the sphygmomanometer at the instant when a faint bruit is first heard on the phonendoscope; it coincides with the moment when the pressure of the armlet *begins* to flatten the circle of the brachial artery. The maximum blood-pressure is indicated by the reading on the sphygmomanometer at the instant when the bruit heard on the phonendoscope ceases; it coincides with the moment at which the lumen of the brachial artery is entirely closed by the pressure of the armlet.

CASE 1.—The patient, a man aged 55 years, had always lived well and been a heavy smoker and taken a good deal of alcohol; he had also been a well-known athlete in his younger days. Twelve months before I saw him, he had had an attack of slight paralysis in the left arm and leg, which laid him up for some time, since when he had had constant attacks of vertigo and palpitation, suffered from insomnia, and his memory was so defective that he had been unable to attend to his business; his condition was gradually getting worse. When I first saw him he looked ill, his lips were somewhat cyanosed, and he suffered from dyspnœa on slight exertion His pulse was 92 to the minute, regular in time, and of small volume (Fig. 1). His vessels felt thickened, but were not very tortuous. His blood-pressure was: minimum, 160 mm. Hg.; maximum, 290 mm. Hg. The area of cardiac dullness was enlarged, extending from the left nipple to the middle line and measuring 4¼ in. across at the nipple level; it would undoubtedly have been larger had he not had marked emphysema, the apex-beat was just outside the left nipple. The sounds at the apex were clear, the first sound being exaggerated; there was a rough murmur, systolic in time, heard over the aortic area. There was no sugar or albumen in the urine. The case was one of arterio-sclerosis and hypertension, with some roughening of the aortic valve and cardiac dilatation and hypertrophy. The patient had a course of "Nauheim" baths, during which I took records of their effect upon the blood-pressure on several occasions. The following table (Table I.) shows the result of the fourteenth bath upon his blood-pressure, which by this time had fallen to 130–240 mm. Hg. The bath was given at a temperature of 94° F., and contained 12 lb. of Droitwich salt, 13 oz. of calcium chloride and half a CO_2 bath.

TABLE I.

	Blood-Pressure.	Pulse-Rate.
Before immersion	140–230 mm. Hg.	62
3 minutes after immersion	115–190 „	64
6 „ „	114–188 „	66
7 „ „	112–188 „	64

It will be noted that the bath has a decided effect upon the minimum as well as on the maximum pressure, and that the pulse-rate was slightly increased. This increase of the pulse-rate is due, I believe, to the necessity of a more rapid action of the heart to fill the dilated vessels when they relax and the pressure first falls; it does not occur in all cases, and even in those in which it is present the rate of the pulse falls to less than it was before the bath, about five or ten minutes after the patient is back in bed. After a course of 25 baths the patient felt and looked very much better; he slept well, and no longer suffered from vertigo or palpitation. The pulse was slower in rate, 63 per minute, and of much better volume (Fig. 2). The area of cardiac dullness extended from ½ in. to the left of the middle line to 1½ in. inside the left nipple, and measured 2½ in. across at the nipple level, a decrease of 1¾ in. The murmur was still present. The blood-pressure had dropped to 120–230 mm. Hg., a fall of 40 mm. Hg. in the minimum and 60 mm. Hg. in the maximum pressure.

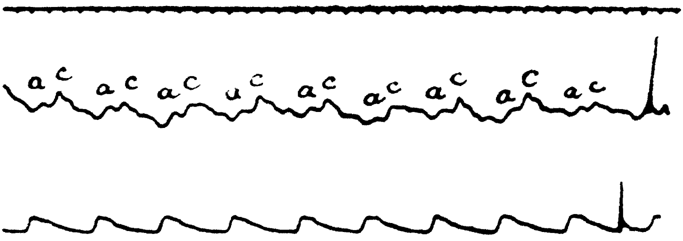

FIG. 1.—*Polygraphic tracing of Case 1 before treatment, showing pulse of small volume, 75 per minute, normal a-c interval.*

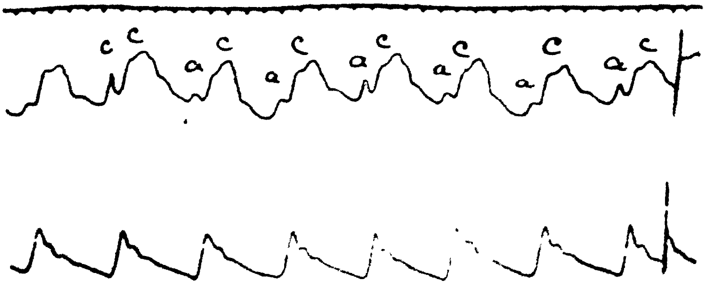

FIG. 2.—*Polygraphic tracing of Case 1, after treatment, showing pulse of improved volume and slower rate, 63 per minute.*

CASE 2.—A man, age 34 years, was interned in Germany for nearly four years, where he only had a yard to exercise in, was half-starved during the last year of his internment, and had to endure much privation in other ways. Early in 1918 he had an attack of influenza, and after this began to suffer from palpitation, exhaustion, and pain over the cardiac area. A German specialist who saw him said he had a dilated and inflamed heart, but no definite treatment was given. When I first saw him he looked thin and ill, his hands were tremulous, and he was in a highly nervous condition; he was unable to sleep without a sedative, and suffered almost constantly from indigestion and severe attacks of palpitation. His pulse was regular in time, rather small in volume, 80 per minute (Fig. 3). His heart sounds were weak, the apex-beat was felt just outside the nipple line, and the area of cardiac dullness was distinctly enlarged, extending from the left nipple to 1 in. to the right of the middle line, and measuring 5½ in. across at the nipple level. His blood-pressure was 90–190 mm. Hg., and there was no indication of any thickening or hardening of the arteries. The case was a typical one of pre-sclerotic hypertension in which the heart had given way under the strain of influenza, privation and anxiety.

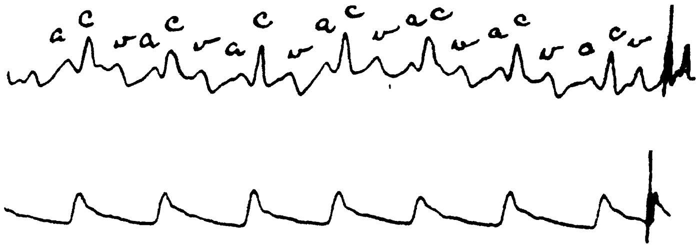

FIG. 3.—*Polygraphic tracing of Case 2, before treatment, showing regular pulse of fair volume, with normal a–c interval.*

Table II. shows the effect of the sixteenth bath upon his blood-pressure, at which time it had fallen to 90–140 mm. Hg. The bath was given at a temperature of 92° F. and contained 14 lb. of Droitwich salt, 15 oz. of calcium chloride, and three-quarters of a CO_2 bath.

TABLE II.

	Blood-Pressure.	Pulse-Rate.
Before immersion - - - -	90–140 mm. Hg.	64
2 minutes after immersion - -	70–130 ,,	68
4 ,, ,, - - -	70–110 ,,	64
8 ,, ,, - - -	75–115 ,,	60
11 ,, ,, - - -	65–120 ,,	60

The patient improved steadily in health, and one month after treatment he had lost all disagreeable symptoms, could sleep well, had no palpitation or indigestion, no tremors or cardiac pain, and his nerves were quite steady. His pulse had dropped from 80 to 60 per minute, the apex-beat was a good inch inside the nipple line, there was no systolic murmur heard at the apex, and the area of cardiac dullness extended from 1¾ in. inside the left nipple to 1 in. to the left of the middle line, and measured 2¼ in. across at the nipple level. The blood-pressure was 90–130 mm. Hg. The systolic murmur heard at the apex before treatment was evidently due to dilatation of the mitral orifice preventing absolute closure of the valves.

Case 3.—The patient, a man aged 47 years, was very obese, his height was 5 ft. 4 ins. and his weight 21 stone. He was cyanosed, suffered from pronounced dyspnœa, and his feet and legs were very œdematous. His pulse was 88 per minute, regular in time, of very small volume, and felt hard (Fig. 4). The jugular tracing was very difficult to get, as the neck was very thick and short and the deep respirations caused constant movement.

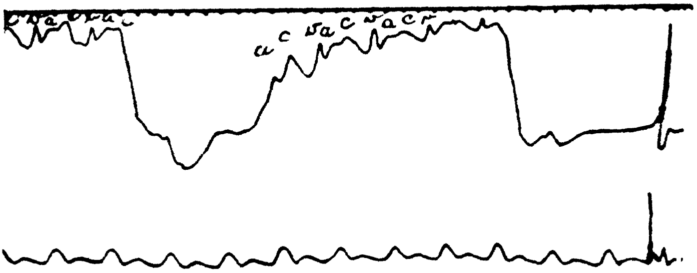

Fig 4.—*Polygraphic tracing of Case 3 before treatment showing pulse of very small volume,* 88 *per minute.*

The area of cardiac dullness was greatly increased, extending from 1 in. inside the left nipple to 2¼ in. to the right of the middle line, and measuring 7 in. across at the nipple level. The heart sounds were fairly clear, and no murmur was present. The blood-pressure was 160 mm. Hg. minimum, and 270 mm Hg. maximum. The case was one of dilated fatty heart. After a course of 25 baths, the patient was of a very much better colour and the œdema and dyspnœa were decidedly less. The pulse was slower in rate, 82 per minute, and very definitely increased in volume (Fig. 5).

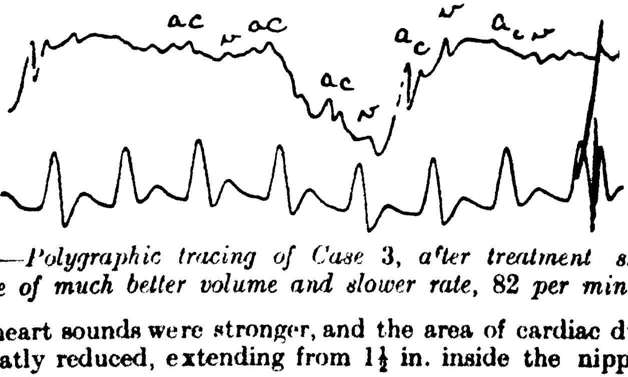

Fig. 5.—*Polygraphic tracing of Case* 3, *after treatment showing pulse of much better volume and slower rate,* 82 *per minute.*

The heart sounds were stronger, and the area of cardiac dullness was greatly reduced, extending from 1¼ in. inside the nipple line

to ¾ in. to the left of the middle line, and measuring 4 in. across at the nipple level, a decrease of 3 in. The blood-pressure was 105 mm. Hg. minimum and 200 mm. Hg. maximum, a drop of 55 mm. Hg. in the minimum reading and 70 mm. Hg. in the maximum reading.

Table III. illustrates the effect of a bath at 93° F., lasting for seven minutes, and containing 9 lb. of NaCl, and 10 oz. of $CaCl_2$.

TABLE III.

	Blood-Pressure.	Pulse-Rate.
Before immersion	150–235 mm. Hg.	100
3 minutes after immersion	100–150 „	108
7 „ „	90–130 „	104

The great drop in the pressure is quite exceptional, a drop of 30–50 mm. Hg. being the average, but it did not inconvenience the patient at all or cause any feeling of discomfort.

Table IV. illustrates the effect of a CO_2 bath at 91° F., lasting 12 minutes, and containing 14 lb. of NaCl, 15 oz. of $CaCl_2$, and three-quarters of a CO_2 bath.

TABLE IV.

	Blood-Pressure.	Pulse-Rate
Before immersion	132–190 mm. Hg.	84
2 minutes after immersion	90–140 „	100
4 „ „	80–130 „	96
6	86–135 „	96
8	85–135 „	92
10	88–132 „	88
12	88–135 „	84

The greatest reduction of the blood-pressure is usually produced by a four to eight minutes immersion, and after this time the pressure usually rises again slightly (Tables IV. and V.).

Table V. illustrates the effects of one of the strongest baths given. The temperature of the bath was 94° F., it lasted 15 minutes, and contained 15 lb. of NaCl, 16 oz. of $CaCl_2$, and three-quarters of a CO_2 bath. Sometimes a whole CO_2 bath is used towards the end of the course, but in this case it was not advisable.

TABLE V.

	Blood-Pressure	Pulse-Rate.
Before immersion	105–200 mm. Hg.	88
2 minutes after immersion	75–130 „	92
4 „ „	70–120 „	92
6 „ „	65–110 „	92
8 „ „	80–116 „	88
10 „ „	82–120 „	86
12 „ ,	80–118 „	82
14 „ „	78–120 „	88

The records in Table III. were made at the patient's eighth bath, when his blood-pressure had already fallen from 160–270 mm. Hg. before the course to 150–235 mm. Hg.

The records in Table V. were taken at the twenty-third bath of

a course of 25, when the pressure had come down to 105–200 mm. Hg. The distinct reduction of the pressure during the course can be seen by comparing these tables. It will be noted that both the minimum and the maximum pressures fell to normal during the baths. This, in my opinion, indicates that the vessels are healthy, and that the case is in the pre-sclerotic stage of hypertension. The behaviour of the blood-pressure in a bath is, therefore, a very valuable aid to diagnosis of the stage of the disease, as well as to prognosis. In Case 1, Table I., neither the maximum nor the minimum blood-pressures fell to normal in the bath; and in Case 4, Tables VI., VII. and VIII., the minimum pressure fell to normal, but not the maximum. This behaviour of the pressure indicates, I believe, that the sclerotic condition has not advanced so far in Case 4 as in Case 1.

Case 4.—The patient, a man aged 71 years, had suffered for several years from dyspnœa and pain in the left side. On exertion or stooping, he complained of headaches, vertigo, and palpitation, and was sometimes awakened at night by choking attacks and a struggle for air. His complexion was somewhat dusky, and he looked drawn and ill. His pulse was 104 per minute and exhibited constant irregularities due to premature ventricular contraction (Fig. 6). It was impossible to obtain the "a" wave in this case, but the character of the polygraph undoubtedly indicated premature ventricular contractions.

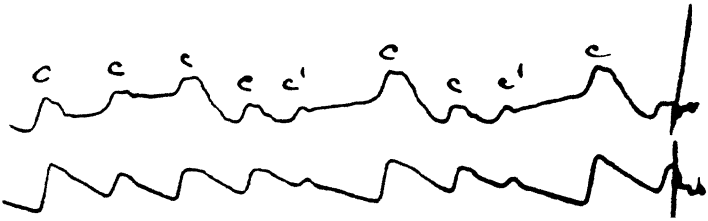

Fig. 6.—*Polygraphic tracing of Case 4, before treatment, showing constant premature ventricular contractions (c). The "a" wave could not be obtained.*

The heart was decidedly dilated, the area of cardiac dullness being greatly enlarged and extending from a ½ in. to the left of the left nipple to 1½ in. to the right of the middle line, and measuring 7 in. across at the nipple level, the apex-beat was in the nipple line. The cardiac sounds were feeble, and a soft systolic murmur was heard at the apex and was conveyed into the axilla. The liver dullness was enlarged, and the stomach was dilated. The legs were markedly œdematous. There was no sugar or albumen in the urine. The blood-pressure was 120–220 mm. Hg. The case was one of fatty, gouty heart, with some arterio-sclerosis. After a course of 25 baths the patient was very greatly improved in health; he was of a good colour, slept and ate well, had no dyspnœa, œdema, or cardiac pain. The pulse was quite regular in time and 60 per minute (Fig. 7).

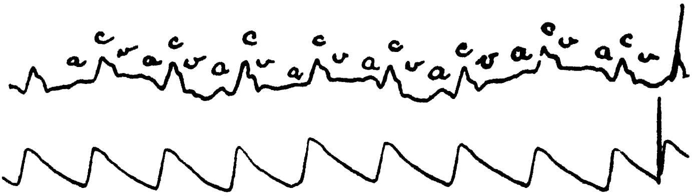

FIG. 7.—*Polygraphic tracing of Case 4 after treatment, showing pulse of improved volume, regular in time,* 60 *per minute.*

The blood-pressure was 90–190 mm. Hg., 30 mm. Hg. less than before treatment. The cardiac sounds were stronger. There was no systolic murmur heard at the apex, this having evidently been due to dilatation of the mitral orifice. The apex-beat was an inch inside the nipple-line and the area of cardiac dullness was almost normal, extending from an inch inside the nipple line to the middle line, and measuring 4 in. across at the level of the nipples, 3 in. less than before treatment. The upper level of the liver dullness was 1¼ in. lower than before treatment. Table VI. illustrates the effect of the sixth bath, which was given at a temperature of 96° F., and contained 8 lb. of NaCl and 9 oz of $CaCl_2$.

TABLE VI.

	Blood-Pressure.	Pulse-Rate.
Before immersion	100–205 mm. Hg.	88
3 minutes after immersion	85–175 ,,	84
5 ,, ,,	88–168 ,,	76

Table VII. illustrates the effect of the sixteenth bath, which was given at a temperature of 94° F., and contained 13 lb. of NaCl, 14 oz. of $CaCl_2$, and half a CO_2 bath.

TABLE VII.

	Blood-Pressure.	Pulse-Rate.
Before immersion	95–190 mm. Hg.	72
2 minutes after immersion	90–184 ,,	78
4 ,, ,,	90–175 ,,	80
7 ,, ,,	78–178 ,,	80
9 ,, ,,	75–175 ,,	72

Table VIII. illustrates the effect of one of the stronger CO_2 baths, given at a temperature of 94° F., and containing 15 lb. of NaCl, 16 oz. of $CaCl_2$, and three-quarters of a CO_2 bath.

TABLE VIII.

	Blood-Pressure.	Pulse-Rate.
Before immersion	90–190 mm. Hg.	68
2 minutes after immersion	85–160 ,,	64
4 ,, ,,	72–160 ,,	68
6 ,, ,,	78–155 ,,	64
8 ,, ,,	78–155 ,,	64

The progressive improvement in the fall of blood-pressure and the slowing of the pulse is well illustrated by the above tables. On his return home his doctor, Dr. Grindlay of Stourbridge, wrote me that the patient appeared to him to be 10 years younger than before treatment.

In cases in which there is definite arterio-sclerosis, the best results are obtained when the temperature of the bath is not lowered below 94° F., as in Cases 1 and 4; but in those in which there is no definite degeneration of the vessels the bath can be given at a lower temperature with advantage (Cases 2 and 4).

CONCLUSIONS.

1. In cases of hypertension, whether sclerotic or pre-sclerotic, the blood-pressure is always lowered by immersion in a "Nauheim" bath, whether that bath is of the *still* or *effervescent* variety.
2. Even an advanced condition of arterio-sclerosis does not counter-indicate the treatment.
3. All cases of hypertension, whether sclerotic or pre-sclerotic, derive great benefit from a properly administered course of "Nauheim" baths.
4. The "Nauheim" baths are useful, not only for the treatment of hypertension and arterio-sclerosis, but cases of sclerotic and pre-sclerotic hypertension can be differentiated by the effect of the baths upon their blood-pressure, for this effect differs materially according to the condition of the vessel walls.

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Malignant Malaria in a Baby—Recovery.

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THE case here reported may be found of interest because of the extreme youth of the patient and the severity of the infection. The child at the height of the illness was moribund, but reacted well to large doses of quinine; she made a perfect recovery, and is to-day none the worse for the attack of malaria.

A girl, aged seven months, came under my care in India on June 29, with the history that for a week she had been fretful and feverish, probably due to teething. On June 30, she cut a lower central incisor, but no relief was noticed in the symptoms of severe general malaise. Two days later (July 2), I noticed that the face was of a waxy colour. The temperature was 99° and the pulse 160. Blood films were prepared from the lobe of the ear, and found to contain a heavy infection of malignant and benign tertian parasites. Hardly a corpuscle was found which did not contain a parasite.

Quinine bi-hydrochloride, gr. 1½, was given four-hourly, and at 10 p.m. an intramuscular injection of gr. 2 in the buttock. On July 3 there was no apparent improvement, the temperature was 103° at noon and the pulse barely perceptible (? 160). Any exertion made the patient cyanosed. The spleen was palpable for the first time, and appeared to be painful when touched.

Treatment.—Brandy ♏5 hourly, tinct. digitalis (♏ 1) and sod. bicarb. (gr. 1), four-hourly. Quinine, gr. 1½ four-hourly, and at 7 p.m. a second intramuscular injection of gr. 2.

The brandy immediately improved the pulse. Food was well taken, and the bowels were moved twice during the day. On July 4, the general condition had improved. The temperature remained about 102° all day. More blood films were made and were found to contain only a single benign tertian parasite, but large numbers of normoblasts were present, showing that regeneration of new red blood-corpuscles was actively taking place

On July 5, the pulse was 140 and the temperature 101°.

The skin acted freely for the first time, and there was not such an extreme degree of pallor noticeable.

Quinine, gr. 1½, was now given t.d.s. and a mixture containing ♏ ½ each of liq. arsenicalis and liq. strych. was substituted for the digitalis. The brandy was continued.

On July 6, the improvement was maintained, and the child was obviously better. There was some gastro-intestinal disturbance due probably to the quinine. I reduced the quinine to gr. ¾ twice a day, and ordered two-hourly feeds of albumen water and whey, and brandy ♏5 every four hours.

It is interesting to note that on this day she recognized her mother for the first time, and smiled several times. From July 7 to 16, she made uninterrupted progress, the temperature remained normal, and her colour improved daily. The quinine was continued until the end of the month (gr. 1 on alternate days) with an iron and arsenic mixture daily.

To summarize, the dosage of quinine was as follows :—

Intramuscular injections (gr. 2), July 2 and 3.
Quin. bi-hcl. gr. 1½, four-hourly, July 2 to 4.
,, ,, T.D.S., July 5.
,, ,, B.D., July 6.
,, gr. ¾, B.D., July 7 to 16.
,, gr. 1, alt. die, until July 31.

There was no relapse, and at the present day the child is perfectly well and healthy.

Practical Notes.

Treatment of Uræmia.

1. *Acute Uræmia.*—With the exception of dieting, treatment has not kept pace with the progress made in the knowledge of the pathology of the affection due to the work of Widal.

Lancereaux's treatment remains as the type of the classical treatment; a drastic purgative, repeated if necessary, a purgative enema, and pills, containing squill, scammony, and digitalis (·05 cg.), eight to ten of which are given on the first day, and again two or three days later. Sweating is promoted by vapour-baths, and, if necessary, cautiously by pilocarpine, 2 cg. by mouth or ½ to 1 cg. hypodermically. Bleeding is done freely.

The actual treatment now in use differs but little from this. Bleeding to 500 cc. is carried out at once, or wet cupping over the kidneys. Lumbar puncture is particularly useful in the nervous forms. Water diet—water sweetened with lactose—is given for four days to an amount of 2 litres if the heart is strong enough, less if not. A saline purge, or a purgative enema, and subcutaneous injections of oxygen are given, as well as diuretics and cardiac tonics such as digitaline and caffein. Renal opotherapy in the form of freshly pulped pigs' kidneys is not free from danger, and the dried powder of sheeps' or pigs' kidneys, though less so, is often quite useless.

Tessier has given intravenous injections of serum from the renal vein with some advantage and without setting up any ill-effects. Isotonic glucose serum (47 per cent.) given hypodermically has been found of service. Pic recommends an intravenous injection of 250 cc. of hypertonic glucose serum (300 per cent.). This has been found useful in acute toxic infections, but has failed in azotæmic nephritis, and even seems to be harmful.

The indications for particular therapeutic measures are furnished by the clinical forms of the affection. In the gastro-intestinal type, vomiting and diarrhœa should be left alone unless persistent, injections of oxygen and purgative enemata are given, and 3 g. of lactic acid in a draught. In the nervous form, bleeding and lumbar puncture (10 to 15 cc.) are necessary, and if convulsions set in, enemata of chloral (1 to 2 g.) or even ½ cg. of morphia. In the respiratory forms, bleeding at once followed by ¼ litre of oxygen, hypodermically, once or twice a day, should be tried, as well as inhalations of oxygen, ether, nitrite of amyl (10 drops), digitalis, and, if necessary, paracentesis thoracis.

2. *Chronic Uræmia.*—In dropsical nephritis, with albuminuria, oliguria, œdema and effusions, the treatment should include digitaline (5 drops), a dechloridizing diuretic, 50 cg. of theobromine a day, to which may be added 25 cg. of benzoate of sodium, or potash salts and vegetable diuretics (corn silk, uva ursi, juniper berries). Hypodermically, oil of camphor and ether

may be necessary. Œdema can be relieved by purgatives, diaphoretics, and Southey's tubes.

The diet should be water for two to four days, and, if no azotæmia is present, the amount of fluids should be reduced. Lactose water or Evian should be used, but no milk whilst the urine is scanty and the œdema considerable. Achard and Widal recommend a chlorine free diet: bread without salt, potatoes, green and dry vegetables, well-cooked eggs, fruit salads, fruit, and milk only for custards and such like. Later, from 2 to 4 g. of salt can be allowed a day. In nephritis with hypertension, dilated left heart, the lesser symptoms of Bright's disease, temporary aphasia, and so forth, hypotensive drugs must be given; nitrite of soda (5 cg.) in teaspoonful draughts three or four times a day, silicate of soda (1 to 3 g. a day), 3 or 4 drops a day of a 1 per cent. alcoholic solution of trinitrin. No iodides must be given. Later, five drops of digitaline for five or six days; squill, adonidine (10 mg. a day). The diet should be milk at first, and then milk and vegetables with occasional days on water diet and purgatives. —(*Journ. de Méd. et de Chir. prat.*, March 10, 1920.)

Importance of Large Doses of Diphtheria Antitoxin, Intra-muscularly and Subcutaneously.

Armand-Delille has found the intra-muscular injection of diphtheria antitoxin, which has been used systematically in the Children's Hospital, to be absorbed quickly and soon bring into action the specific effect of the serum, thus constituting a real progress in treatment. It has, however, one drawback in that the serum is eliminated rather rapidly, which he considers is the probable explanation of the later symptoms of intoxication, particularly paralysis, which have been noticed in a series of cases treated in this way. He has, consequently, followed up the intra-muscular injection by one the next day, given subcutaneously. As a result of this he has only observed a very small proportion of cases develop paralysis subsequently—five times less than when the intra-muscular injection only has been given. He believes that this association is certainly the most efficacious method of treatment. The absorption and elimination of the diphtheria antitoxin, when injected subcutaneously, is very much slower than in the case of intra-muscular injections, and thus a continuous supply of the antitoxin is kept up to the organism after the effects of the initial intra-muscular injection have passed off. He has adopted the procedure of giving to children up to 10 years of age from 30 to 40 cc. by intra-muscular injection on admission, and from 40 to 60 cc. by hypodermic injection on the following day. In the case of very toxic or malignant throats these doses are increased, and if on the third day membrane still persists, another hypodermic injection of 30 to 60 cc. is given. In this way the rapid effect of the intra-musuclar injection is secured, and the slower effect of the subcutaneous injection completes the treatment most satisfactorily. —(*Le Bulletin Médical*, March 17, 1920.)

Reviews of Books.

Electrical Treatment. By WILFRED HARRIS, M.D., F.R.C.P. Third edition. London: Cassell & Co., Ltd.

A PERUSAL of the third edition of this well-known work serves to remind us of the very large amount of satisfactory work which can be done with no other instruments than a galvanic battery and a Faradic coil. The introduction within the past 20 years of treatment by high frequency, X-rays, and diathemy has tended to obscure the work done by the early electro-therapeutists, whose armamentarium was for the most part limited to the two simple pieces of apparatus above mentioned, with the addition, perhaps, of an inefficient static machine. Yet it is a fact that in many instances their results were at least equal to any obtained now.

The author, in view of his own speciality, naturally devotes a large part of the book to the diagnosis and treatment of nervous diseases. As to diagnosis, the reviewer has been unable to find any reference to the use of condenser discharges. This is strange, for, even though it be admitted that their employment has not revealed anything strikingly new, they at least provide a means of using an interrupted current in which impulses of known length can be used at a known voltage and at a definite rate per second. This enables tests to be repeated with an exactitude impossible with a Faradic coil, and hence permits of accurate records.

Speaking of infantile paralysis on page 142 the author seems to imply that muscles which will not respond to the Faradic currents are very unlikely to recover. In the reviewer's experience, unless a muscle is actually replaced by fibrous tissues, it can never be safely prophesied that it will not recover, provided that it be given rest in a relaxed position. Much unnecessary grief is caused to parents by too-confident predictions of evil based solely upon electrical examinations, no matter how carefully these are carried out.

Careful directions for treatment are given for a great number of conditions, and one is glad to see sinusoidal currents advocated for spastic paraplegia by a specialist in nervous diseases, as therapeutic nihilism is the usual attitude of the professional neurologist.

No mention is made of the use of X-rays in disseminated sclerosis, nor of the high frequency condenser electrode applied *per rectum* in vesical and rectal incontinence associated with chronic progressive diseases of the spinal cord. This seems a pity, for such measures, if the benefits they produce are not permanent, appear to be at least as valuable as, *e.g.*, the use of X-rays in leukæmia.

This book may be read with profit by anyone in active practice, no matter what his particular speciality may be.

Mind and its Disorders. By W. H. B. STODDART, M.D., F.R.C.P. Third edition, with illustrations. London: H. K. Lewis & Co., Ltd. Pp. 580. 18s. net.

THIS, the third edition of Dr. Stoddart's text-book, differs materially from the former editions owing to the fact that the author has, he tells us, since writing the last edition changed his attitude towards mental disease by adopting the doctrines of Freud. One of the results is that the book contains a considerable amount of controversial matter about which in many instances one cannot help thinking it would, from the point of view of a text-book, have been wiser to have been less dogmatic. While the book contains some interesting descriptions of the different mental diseases, there are parts in which the accounts are somewhat contradictory, and one gets the impression that the author has occasionally found it a little difficult to make all his facts correspond with the views to which he has become so completely converted.

An Atlas of Dental Extractions. By C. E. WALLIS, L.R.C.P., M.R.C.S., L.D.S. Pp. 26, eleven plates. London: J. and A. Churchill. 6s. net.

THE general practitioner of medicine, in remote country districts and in the poorer areas of great cities; the naval or military surgeon; the medical officer in colonial districts all need an adequate knowledge of the methods of extracting teeth. In this little monograph, now in its second edition, a practical and well-illustrated account of this branch of minor surgery will be found. The title scarcely does justice to the character of the book, for, in addition to the description of the extraction of the teeth, a short summary of the diagnosis of the common causes of dental pain, with directions for simple remedies, is included. There is also an account of the use of local anæsthetics for dental purposes.

The plates illustrating the operation of extraction give the form of the tooth, a representation of the appropriate forceps for each of the several teeth, and one or more photographs conveying an appropriate idea of the proper stance for the operator and the methods of holding the forceps, supporting the jaw and preventing undue injury to the soft parts. We may comment upon the garb of the operator in these illustrations, for it does not convey the importance which is now attached to asepsis in dental operations. We also note that the writer does not emphasize the importance of the habitual prescription of a mouth bath after extractions have been performed, and in our experience medical practitioners are often remiss in this important respect.

In conclusion, we may confidently recommend the book as a straightforward and practical description of dental conditions.

Preparations, Inventions, Etc.

"BAUME DURET."

(London : The Anglo-French Drug Co., Ltd., 238A, Gray's Inn Road, W.C.1.)

This preparation was devised by Dr. Giffon, and has been in use at the St. Louis Hospital in Paris for some time. The formula was communicated to the *Société française de Dermatologie* at the request of Brocq, Danlos, Hallopeau and other physicians to that hospital. Its active principles are tar, camphor, and sulphur dissolved in acetone, combined with oil of cade, borax, resorcin, guaiacol, and menthol. It is semi-liquid in consistency, has a camphoraceous odour and a yellowish colour. The stain caused on the skin and on linen is not permanent. Sulphur, when dissolved in acetone, loses its irritant properties, and the use of this preparation has yielded very satisfactory results in cases of eczema, impetigo, acne, sycosis seborrhœa and prurigo; it may be used upon children without fear of irritating effects being set up.

"CURATOXINE VENDEL."

(London : The Anglo-French Drug Co., Ltd., 238A, Gray's Inn Road, W.C.1.)

This preparation is composed of iodated tormentilla, glycerophosphates, vegetable extracts having laxative, diuretic, and sudorific properties, and a combination of hexamine, sodium benzoate, and lithia, known as urolithine.

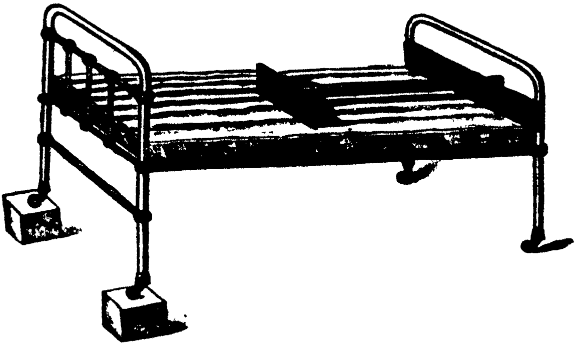
It is in the form of brown, soluble, saccharated granules, which dissolve easily in the mouth and have an agreeable flavour.

It has been introduced for the internal treatment of chronic dermatoses, caused by animal and vegetable parasites, and by pyogenic micro-organisms, such as furunculosis, folliculitis, eczema impetigo, and acne.

CUTHBERT'S PROP.

(London : Messrs. Allen & Hanburys, Ltd., 48, Wigmore Street, W.1.)

This prop has been devised by Mr. Firmin Cuthbert to retain patients in the Fowler position. The patient's buttocks rest against the narrow shelf, which is covered by a pillow. Each leg rests on a pillow placed along each side of the vertical bar, and the position of the shelf is adjusted according to the length of the patient, and fixed by a peg running through the bar. The shelf is easily adjusted and slipped down when necessary attention to



the pelvis is required, it is easy to clean, and it is inexpensive.

COMBINED PRIVATE AND PROFESSIONAL ACCOUNT BOOK FOR DOCTORS.

(Leicester : Messrs. J. McQueen & Co., Moat Road.)

This book has been arranged by the designer of the A.B.C. System of Accounts for Business. The main point is that it furnishes a professional profit and loss account which, it is stated, will satisfy the most exacting surveyor of taxes. The system is most comprehensive and, at the same time, very concise and intelligible. It will be found to simplify very materially the terrible drudgery of book-keeping.

SEDOBROL "ROCHE."

(London : The Hoffman-La Roche Chemical Works, Ltd.,
7 and 8, Idol Lane, E.C.3.)

This preparation is now put up in the form of "soup tablets," each of which contains 17 grains of sodium bromide. One or two of these dissolved in a cupful of hot water make a broth, which is seasoned so as to have an appetizing aroma and a pleasant palatable flavour. The presence of the bromide supplies the necessary amount of saltness, but not too much so, being thoroughly well masked, so that the patient, in drinking a cup of this bouillon thus prepared, has no suspicion that a dose of bromide is being given at the same time.

The tablets are issued in airtight boxes each containing ten

THE PRACTITIONER

JUNE
1920

The Position of the General Physician in Medicine.

BY SIR BRUCE BRUCE-PORTER,
K.B.E., C.M.G., M.D., ETC.
Late Commanding the 3rd London General Hospital, etc.

I HAVE used the word "Physician" rather than "General Practitioner," for the latter is essentially a man who is obliged, by his position of residence in the country, to practise all that is included in the words—Medicine, Surgery, and Midwifery. The progress of surgery since the introduction of anæsthetics and asepsis has been enormous, and, in many cases, dramatic. The progress of medicine during the professional life of most of us has been, if anything, greater than that of surgery. The time has now arrived when it is impossible for any single physician to keep absolutely up to date in every branch of medicine, and specialism is essential. The more specialized medicine becomes the more important the position of the general physician, who thus becomes a most important specialist.

I would define the general physician as the man who has a general knowledge of medicine, and who realizes his limitations. One of the most important things for a general physician to know is, when he does *not* know. By this, I mean that, having decided which system is at fault, he should be able to admit that help is to be gained by calling in a specialist. I do not suggest for a moment that every organ of the body requires a specialist.

I use the term specialist to mean those men who practise surgery, gynæcology, neurology, radiography, bacteriology, pathology, ophthalmology, and subjects of this type. Affections of the heart, lungs, and abdominal organs are, to my mind, all in the province of the general physician, for he is much better qualified to diagnose and treat those organs than a man who confines his study to one of them only. Specialism which divides the body beyond a certain amount, such as I suggest, tends to approach the medicine of Ancient Egypt, where the body was so divided that there was a doctor for every organ, and the doctor for one organ was not allowed, at any time, to treat another.

Whilst realizing that each branch must co-ordinate its work with the others, I would like to divide medicine as it appears to me into: (1) Preventative; (2) Diagnostic; (3) Curative.

Preventative Medicine has been demonstrated in the highest degree in the recent war. We have seen war carried on in countries, which had hitherto been death-traps for armies, with a smaller death-rate from disease than that prevailing at the same time among the civil population at home. The field of *Preventative Medicine* is mainly that of the public health officer, but he in turn must depend upon the next branch, namely, that of the *Diagnostician*, for early notification of disease of a preventible and infectious nature. The *Curative* branch is to my mind, strange as it may seem, the least important of the three. Many medical ailments at present get well in spite of the physician and his drugs, and we have all lived long enough in medicine to see many vaunted specifics proved of no value. The most important thing is correct and early diagnosis, and we recognize that many serious illnesses would be avoided by earlier recognition of the symptoms.

The public is to blame for much that it suffers at the hands of our profession. It values the services of the physician too lightly, and, while it would never dream of trying to regulate its own or its neighbour's watches, it practically never consults a doctor until its own idea of treatment has been tried and has failed. If any ordinary man bought a property, he would not risk a faulty lease by dealing with it himself, or by asking a non-professional man to do so for him; yet the same man will risk the lease of his life by allowing his non-medical friends to diagnose and treat his complaint.

Again, the ignorance of the public in matters relating to health is very great, and the doctor is expected to make a diagnosis and to treat the case in the penny-in-the-slot fashion. The public cannot realize that a small thing, such as a cough, may be due to many causes, and that the early and correct recognition of the cause may call for the help of a group of specialists (*a*) the throat specialist to exclude all sorts of causes from Singer's nodules to early malignant; (*b*) the radiographer to show the movement of the diaphragm, and to exclude the presence of early or old tubercle in the lungs; (*c*) the bacteriologist to examine the sputum for T.B. or other organisms. The ordinary patient expects to walk into the physician's consulting room, and come out in a few minutes with some hieroglyphics on paper guaranteed to cure. The length of time allowed is not much more than the Chinese medicine man requires to shake the sticks out of his box. Again, the average patient does not consult a doctor until something has gone wrong with the works, and then he consults someone who has been recommended to him by a chance friend or acquaintance. The doctor has never seen him before, during his normal state of health, so cannot possibly say how far he has departed from it.

The training of the doctor is carried out in hospital where various specialists assist in the diagnosis, and he is then let loose on the world to make his diagnosis practically on his own. The strain of building up the reputation of a sound physician is greater than that of building up any other career, and the young man is often afraid to suggest another opinion from a mistaken idea that by so doing he will admit a lack of knowledge that would do him harm. I am sure, if we could educate the young medical man and the public as to the reasons for second opinions, we should render a great service to both. In legal questions, counsel's opinion is sought in all difficult cases, and often several judges will consult and reserve their judgement for a long time before giving their opinion, yet no legal decision can be so important as those which we have to make, affecting as they do, matters of actual life and death. In theory "all that a man hath will he give for his life."

The patient often wishes to have a second opinion, but is afraid to say so, lest he should offend his doctor, and so goes off to all sorts of specialists. It is a dangerous way of seeking help, for each specialist is bound to have his opinion tinged by his specialism. I have recently seen an old patient of mine, who suffers from nervous exhaustion; before the war I had been able to direct his steps, when worried by subjective symptoms, to men whom I knew would listen to what I could tell them about his history. The war took me away on military service, and in my absence he started on a course of consultations with specialists. He had his nose operated upon, underwent two abdominal operations (the last being the removal of a large part of the colon), treatment by an osteopath, and a course of treatment abroad by a specialist in ductless glands. He is now, on the advice of a specialist in Paris, going to Plombières for treatment. I am wondering what is expected from Plombières douches in the case of a patient who has lost most of his large intestine. In fact, the man is a walking encyclopedia of specialists. The habit of consulting a specialist has become with him a vice. Every man at forty is said to be either a fool or a physician, so I leave the reader to decide the category in which we would place him. This man would have escaped much, had he fallen into the hands of a general physician at the end of 1914.

I can quite understand that many men think patients will object to being passed on to have a number of opinions before the general physician is in a position to give his diagnosis, but I have now been in practice for many years, and I am sure that this is not the case. The total cost in fees will come to much less in the end than a number of visits in which symptoms only are treated, and the result is much more satisfactory.

During the war I have been in command of hospitals at home and in Mesopotamia. In our big military hospitals, through which millions of men have passed, the public has become used to having a number of specialists called in to examine the cases. In the scheme of the Ministry of Health, it is contemplated giving the doctors on the panel system the assistance of hospitals and teams of specialists. This addition to the panel system will create a body of general physicians among the panel practitioners. There will always be left in England, no matter how perfect a State Medical Service may be evolved, a large mass of people who will prefer to choose their own doctors. The personal element, which counts for so much in our relations with our patients, will ensure this, and it is on this account that I wish to accentuate the importance of the part of the general physician in the near future. State Medical Service will never satisfy the individualist. He will chafe at the red tape and routine which are inseparable from State Service, and so, while young men may, when first qualified, seek State medical work, the more ambitious will leave it for private practice later on.

It is the more important, therefore, that those of us, who have established positions in the medical and social life of the country, should do propaganda work among the public as well as among the profession, so that a special group of general physicians may arise in all our big centres; men to whom the public can turn for advice, knowing it will get the full benefit of every specialist who can help in forming a diagnosis.

I have followed this line of work for many years, being the type of work that appealed to me. I have had one or two names given to me, which were intended to be facetious, but I thought them excellent. One was "a specialist in specialists," and the other "an editor of the opinion of specialists." I believe we should try to put ourselves in the position of the patient a little more than is usual in our profession. We seek specialists for ourselves when we have symptoms that worry us, and we do not consider it odd that we seek a throat man for an irritable throat or cough, or use the radiographer for a doubtful abdominal attack.

I have reason to be more than grateful for the assistance I have had in my medical career from all sorts of specialists, and I have never regretted advising my patients to allow me to share the responsibility with a colleague who has made a special study of the system at fault. It proved also to be an excellent way of acquiring the latest knowledge in special subjects, whether of the newly recognized diseases or of the new forms of treatment.

The advice I would give to the public is to treat its own engine at least as well as it does its automobile, *i.e.*, do not wait until it breaks down before having it overhauled. Select a good general physician and get him to examine you when you feel at your very best; let him make his notes on your condition, and, if no defects are found, go on your way, reporting again in six months. If illness comes, the physician will know how far you have departed from your normal. Should he find trouble of a nature outside the province of a general physician, let him call in the men whom he knows to have made a special study of that system. He will then be in a position, having collected all the evidence, to make a really correct diagnosis as far as such a thing is humanly possible. Only when this general examination becomes a routine measure in the life of the public, shall we get to the ideal position of detecting disease at the very beginning, and so of curing diseases which are often incurable when patients come to consult us now.

To the general physician, my advice is to get as good a general knowledge of disease as you can, but do not try to get the intimate knowledge of special subjects (such as those I have named), that specialists in those subjects must possess. There will always be more than enough work for us all, if we are only agreeable and do our work honestly, and the course I advocate will be for the good of the patient, which, after all, is the main reason for our existence.

The physician of to-day has taken the place of the priest of former days, and often the patient comes with some trifling ailment because he or she wants the advice of the man of the world, and this side of the work is essentially that of the general physician, who thus becomes the trusted friend.

I feel that I have even now but touched in a most sketchy way the fringe of a very big subject. If, in the future, the general physicians can be persuaded to collect the records of their practices, we should get something we do not at present possess, *i.e.*, a good work on prognosis in diseases when they occur among the non-hospital patients. I would like now to give a few typical cases in which special help was of vital importance to the patient :—

(*a*) A patient came to me complaining of pain in her wrists. She looked a healthy woman of middle-age, and complained of nothing else. A thorough examination revealed a to-and-fro murmur behind the upper part of the sternum, and an X-ray examination by Dr. Howard Humphris showed a pulsating swelling of the aorta, about the size of a tangerine.

(*b*) A man came to me complaining of a pain in his chest, which occurred on waking in the morning, lasted a few seconds, and came on, sometimes twice, in the forenoon. It was not produced or affected by food or exertion. He had been treated by various doctors for dyspepsia, but an X-ray showed a small aneurism, He was a deep-chested man, and no murmur could be heard unless he lay on a couch with his chest in a condition of forced expiration. and then a faint to-and-fro murmur could be heard.

(*c*) A patient complained of tightness of his stomach, which might easily have passed for dyspepsia, as he did himself too well, but showed on thorough examination an absence of pupil reflex, and of ankle jerk, but knee jerk was present. There was no history of syphilis, but various negative Wassermann blood tests. My friend, Sir James Purves Stewart, did a lumbar puncture, and the fluid was positive, and showed large increase in lymphocytes.

(*d*) In a case of supposed bromism, I found that the patient was in a condition of partial coma.

I could give many similar cases, but have given enough to illustrate my remarks and know that everyone can add to the examples.

Compression Neuritis due to the Normal First Dorsal Rib.

BY SIR W. I. DE C. WHEELER, M.D., F.R.C.S.I.

Formerly Lt.-Col. R.A.M.C. and Hon. Surgeon to H M. Forces in Ireland; Surgeon, Mercers' Hospital, Dublin.

TWO cases of compression neuritis, under the care of Sir Harold Stiles, one of which I saw in Edinburgh six years ago, stimulated my interest in a condition which, until recently, has escaped general observation. At first sight the cases appeared to be some form of ulnar nerve neuritis, occurring in adults without trauma, or other obvious reason. On consideration it would have occurred to a surgeon that these patients were suffering from the presence of a cervical rib, but X-ray examination gave a negative result.

During the last few years I saw two cases which I believed to be in every respect similar, but my suggestion that the condition might be relieved by operation was at variance with the opinion of those who had been previously consulted, and I was left with a polite indication from the patients that the activities of surgeons should be placed under some form of legal restraint.

About 12 months ago an athletic clergyman, 35 years of age, presented himself with the following history. He had met with no injury, other than the commonplace falls at football, etc. He had no illnesses worthy of note, except influenza, but he had noticed for the past four years a gradually increasing loss of grasping power in his right hand. The loss of power began with painful cramps, which he attributed to too much writing. Improvement sometimes followed treatment, then a relapse would occur, and he was worse than before. On waking in the morning the arm would "fall asleep," writing became impossible, and he noticed a gradual wasting of the muscles of the hand. He thought that the first finger and thumb were the worst at first, but subsequently the last three fingers were the most affected. Various doctors were consulted, and he was treated, *inter alia*, for writer's cramp, neuritis, and syringomyelia. He always carried a specially warm glove to protect the affected hand from cold.

On examination it was found that the interossei muscles were practically non-existent, and that there was weakness in the median distribution, as shown by the poor attempts to flex the first finger. An inability to extend the wrist fully without flexing the fingers, pointed to involvement of the musculo-spiral nerve. Generally speaking, the appearance of the hand suggested a mild variety of *main en griffe*. Loss of power was most pronounced in the area supplied by the ulnar nerve. Sensation appeared normal, except in the ring and little finger, and here it was only slightly altered. He could not tell degrees of hot and cold with the same facility as when the test was applied to the normal hand, and he could appreciate light touches better than pinpricks. Pain, but for the initial cramps some years previously, had been conspicuous by its absence, and in this respect his case differed from others recorded. X-ray photographs demonstrated the absence of cervical rib, and showed a normal thoracic inlet.

During the examination the patient made a statement of great interest, with direct bearing on his condition. He said that the gripping power of his fingers depended greatly on the position of the arm. Above his head his power was increased, and he could write on a blackboard steadily, and with ease. Grasping a tea-cup, on the other hand, was almost impossible. On giving him a pen and paper he wrote his name like a man suffering from paralysis agitans; but when the paper was put on the wall above his head the writing was clear and steady.

Treatment by massage, ionization, hot-air baths, etc., was tried for several months. Special attention was given to the upper fibres of the trapezius, in the hope of increasing the support of the pectoral girdle upwards. There was no lasting improvement in his condition, and a paper published in the *British Journal of Surgery*, by Stopford and Telford, in October 1919, describing ten consecutive cases of the condition, removed any remaining doubts as to the best line of treatment to adopt.

The operation of removal of the first rib is out of the beaten track of most surgeons, and a lively recollection of the anatomy involved called for pause. The experience of this case, however, and subsequent operations on the cadaver leads me to think that the operation is not difficult, and may be accomplished with practically no risk.

On January 1, an incision was made above the clavicle, as if for ligature of the subclavian artery, a second limb was added, running parallel to the fibres of the trapezius muscle. A little dissection brought the external jugular vein, the posterior belly of the omo-hyoid, and the nerves of the brachial plexus, crossed by the transversalis coli vein into view. A finger in the wound found the back part of the first rib much higher in the neck than was mentally assumed. The brachial nerves were drawn forwards and downwards, and the interval between the scalenus medius and levator anguli scapulæ muscles defined. The roots of the nerve of Bell in this position were not seen, but their hidden presence was respected. The attachment of the scalenus medius muscle to the first rib was divided sub-periosteally, and the inner border of the bone cleared of periosteum and Sibson's fascia. Doyen's rib elevator and cleft-palate raspatories were used to bare the underside of the rib, and to separate it from the underlying pleura.

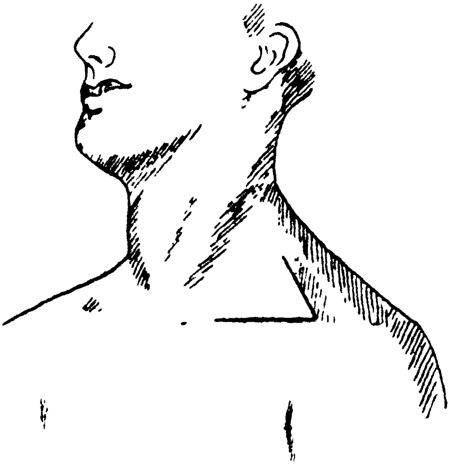


FIG. 1.—*Incision for removal of the first rib working from behind.*

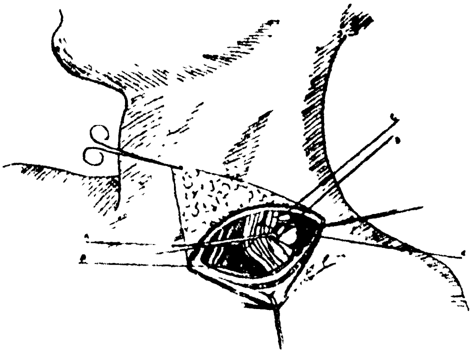


FIG. 2.—*Retraction of plexus forwards towards scalenus anticus* (A) ; *Artery* (B) ; *Trapezius and Levator anguli scapulæ* (E).

The rib was then divided with shears behind the scalenus medius. I felt I could not get far enough forward by dissection from behind, and when the rib was defined I retracted the nerves and other structures back, and cleared the bone between the artery and the lower brachial trunk. The rib was now cut in front of the groove, which is supposed to carry the subclavian artery. It was noted that the artery did not lie in direct contact with the rib, but the nerve was tightly stretched like a band across the inner border. At the completion of the operation, when cutting away an irregular point of bone, either the external jugular or subclavian vein was injured. The bleeding was controlled in a moment, and we had the satisfaction of feeling that the bone had been divided well in front of the compression point. The segment of rib removed extended from the posterior edge of the insertion of the scalenus medius to the scalene tubercle, so as to include the groove for the subclavian artery, and the trunk formed by the eighth cervical and first dorsal nerves. Subperiosteal resection of the first rib may possibly be followed by regeneration of bone, but there are two reasons why a recurrence of compression of the nerve is unlikely :—1st. After removal of the segment at fault the divided ends of the rib fall towards the thorax, and new bone would take a curved direction away from the plexus. 2nd. The periosteum is of necessity stripped from the rib like rolling up a sleeve, and

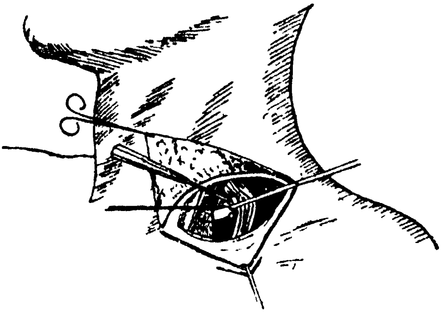


FIG. 3.—*Exposure of the rib nearer the compression point, the plexus is now retracted backwards.*

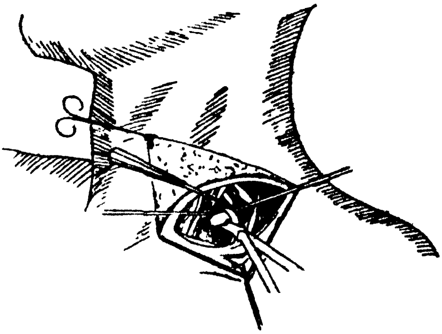


FIG. 4.—*Division of rib between the retracted artery in front and the plexus behind.*

not by two flaps in the manner usually adopted lower down.

Some months must elapse before the result of this procedure can be judged. Following operation, as might be expected, there was more pronounced sensory and motor disturbance over a wide area, due to the retraction and handling of the plexus. In Thorburn's cases of cervical rib, pain was relieved by operation in 80 per cent.; paralysis was cured in 50 per cent.; impairment of sensation was improved in all, but cured in none; wasting usually persisted.

In addition to the cases operated upon by Stiles (unpublished), Thorburn, Murphy, Morley, Stopford, and Telford have drawn attention to brachial compression neuritis produced by a normal first rib.

The symptoms in a general way are those of cervical rib. Pain along the ulnar border of the arm and forearm was present in all the cases described by Stopford. The pain was always increased by anything producing depression of the shoulder girdle. He states that atrophy and paresis affect for the most part the intrinsic muscles of the hand, but the flexors and extensors of the wrist not infrequently are involved. Loss of protopathic sensibility is greater than the epicritic loss; a dissociation which denotes nerve compression.

Ætiology.—Any condition which lowers the shoulder girdle, such as fractured clavicle, will pre-dispose to compression by bringing the lower trunk in more close relationship with the first rib. The majority of cases, however, occur in young adults without apparent cause. There is some evidence to show that when an unusually large contribution from the first or second dorsal nerves goes to the formation of the lower trunk of the brachial plexus, any condition which lowers the shoulder girdle may cause compression against the first rib. At puberty a descent of the shoulder normally takes place, especially in women. If there is muscular weakness, and this normal descent is exaggerated, and, at the same time the lower brachial nerve trunk contains a larger contribution than usual, compression is likely to occur.

Diagnosis.—The history of the case; pain along the internal cutaneous nerves; atrophy of the intrinsic muscles of the hand; the perception of touches with cotton wool, with the absence of distinct feeling of pin-pricks to the inner fingers; and very constantly a loss of accurate differentiation between degrees of hot and cold are guides in excluding rheumatism, or simple neuritis. Inflammatory conditions of the nerve roots (radiculitis) are often associated with inequality of the pupils, due to sympathetic involvement in a position above the point where compression of the lower trunk takes place at the first rib. Syringomyelia must be excluded; and it is interesting to note that Murphy has drawn attention to an association between this disease and the presence of cervical rib. J. B. Murphy states that occasionally elongation of the transverse process of the seventh cervical vertebra, or acute angulation of the first rib at its juncture with the transverse process, produces the same symptoms as a true seventh cervical rib. Great care must be taken to ascertain, before removal of a cervical rib, that it is the real cause of the trouble. At least one case is described in which a cervical rib was present, but the compression of the nerve was due to a normal first rib, and the symptoms disappeared after removal of the latter.

I have to thank Dr. A. K. Henry for his assistance in connection with the literature on the subject, and for valuable suggestions made when working on the cadaver. Dr. McAuley kindly assisted me at the operation, and Sir Harold Stiles wrote me a full description of the operation as performed by him.

Medico-Legal Notes

(*concluded*).

BY SIR JOHN COLLIE, C.M.G., M.D., J.P.

Medical Examiner to the London County Council; late Director of Medical Services, Ministry of Pensions; Home Office Medical Referee, Workmen's Compensation Acts, etc.

RECOGNITION that it is the driving force of routine which keeps most of us at the point of duty, when we would rather be elsewhere, should help us to realize how difficult it is for working men, whose education is incomplete and whose perspective is blurred when dealing with themselves, to take their courage in both hands and return to work at a time when their muscles have become atonic and the work-habit is broken. It is a diagnostic point of great subtlety to decide where the professional valetudinarian ends and the malingerer begins. The mental impression of invalidism resulting from months of introspection is a very real one. Sub-conscious cerebration has more to do with influencing character and conduct than we sometimes give it credit for.

The incapacity of most working men to appreciate ordinary moral obligations, when it rests with them to decide whether they shall or shall not return to work after an injury, seems to be almost congenital. I have seen an immense number of men who, commencing with a sturdy independence and an honest desire to return to work when able, have gradually become mentally and morally debased, solely as the result of idleness brought on by delayed litigation under the Workmen's Compensation Act. Even though the wolf is at the door, the sense of responsibility seems to be obliterated; but moral responsibility, even amongst the highly-educated, is a variable quantity.

Dr. Byrom Bramwell defined a malingerer as one who "feigns sickness, or who deliberately (knowingly and wilfully) induces or protracts an illness, with the object of avoiding duty, claiming money compensation, exciting sympathy, or for any other reason." Many working men are liable to exaggerate symptoms if, by doing so, pecuniary advantage accrues to them.

Pain in the back following an accident is a condition which is often caused by auto-suggestion; but the complaint of pain is not necessarily malingering, though the pain is often only psychic. The pain of a neurotic satisfies an unconscious longing for something which will command sympathy, and it is often profitable. Psychologists tell us that we conceive only that partial aspect of a thing which the individual regards, for *his* purpose, as its essential aspect. For instance, the substance chalk is looked upon by different people according to the use to which it is put; the geologist thinks of it as the cemetery of millions of animalcula; the schoolmaster, as a messy but useful aid to imparting knowledge; the chemist, as carbonate of calcium. In short, the essential quality of a thing is its worth to the individual, and its value to him is its power to serve his private ends. On one occasion, when examining a working man for an injury to his thumb, he observed me examining the terminal phalanx of one of his fingers, which had been partly removed, obviously as the result of a former accident. "That," said he, "is of no importance; it was done at home"!

Surprise visits are often very useful in suspected cases of malingering, and are sometimes more than justified. On one occasion, I was detained a suspiciously long time before admission to the house of a working man, who was alleged to have had an accident. I heard a good deal of noise going on inside the house, and, thinking that the patient had probably been getting into bed whilst I waited, I ventured, after I had examined him in bed, to look below the table, which was covered with an overhanging cloth, and there found his clothes, which he had evidently just taken off! I have no doubt that, had I followed the example of the late Mr. Rose, and put my hand into his boots, I should have found them warm.

A. E., who for some time had been in regular steady employment in the lead trade, upon the declaration of a strike, immediately complained that he was suffering from lead-poisoning, and consequent paralysis of the right arm, and furnished medical certificates to that effect. As the case gave rise to some suspicion, it was referred to a medical man, who fearlessly reported that the case was one of malingering; but his opinion was not acted upon, and A. E. for many months was paid compensation for right-sided "drop-wrist." He appears to have been no mean actor, for whilst he simulated paralysis of the right wrist so well as to obtain weekly certificates of incapacity, he was actually engaged in giving performances as a professional strong man. Every night at a music-hall, clad only in gorgeous harness, he lifted enormous weights. One of his demonstrations consisted in stiffening the muscles of the *right* forearm and wrist, and breaking with his right hand a thick strap which passed diagonally over from his right shoulder to his left hip!

When detected, he not only admitted the fact, but boasted of how he had hoodwinked the examining medical men, and offered, for the payment of £5, to appear as a witness against another man who was also receiving fraudulently compensation for alleged lead-poisoning. He had the temerity to leave with his employer some picture postcards, which I now have in my possession, showing himself in his professional garb and attitudes. He volunteered the information that these photographs were taken at the very time when medical men were examining him and certifying that he was at his worst!

It is very difficult for a medical man, who knows what serious results often follow trivial accidents, to believe that anyone, however impecunious or depraved, would willingly court an accident; yet I am satisfied that a small proportion of so-called accidents are deliberately self-induced. Recently I investigated a case for a railway company in which a man had, within ten years, claimed damages for eight accidents and obtained sums varying from £100 to £500 for each. He was astute enough to change his name occasionally. As a sequel to the last of his alleged accidents (of which there was no proof), he feigned dementia and kept it up for *a whole year!* After my examination I strongly advised the railway company to fight the case, and stated I was prepared to go into court and prove that his so-called mental disease was a sham; but his wife settled the case by refusing to give evidence, as she would not face the witness box and the cross-examination she knew she would be subjected to.

The following story illustrates nothing in particular. It is an example of malingering *par excellence.* It will not edify, but may perhaps amuse.

The vicar of a very poor London parish, well known for his practical but discriminating charity, made it a rule personally to visit all cases before giving assistance. Late one Christmas Eve, when his family were entertaining friends, a piteous message was sent to him, to the effect that one of his parishioners had died, that there was no food in the house, and that the widow was at the door begging for a few shillings. The clergyman, urged by his family not to leave the house on such an occasion in such weather, for a storm was at its height, for once hesitated; but eventually interviewed the woman, and followed her to a tenement house, the dark creaking stairs of which he ascended. When he entered the death chamber, he saw lying on the bed the form of the breadwinner of the family, reverently covered with a white sheet After some conversation with the widow, and writing out various soup and coal tickets, he took his departure. When halfway down the stairs he discovered that he had forgotten his umbrella and rapidly returned, when, to his astonishment, the "corpse" was sitting up in bed chortling!

Medical Notes.

(Second Series)

(*continued*).

By SIR THOMAS HORDER, M.D., F.R.C.P.

Physician, with charge of Out-patients, to St. Bartholomew's Hospital, etc.

DISEASES OF THE KIDNEY.

PACE physiology, one is tempted to say that there is no such thing as "normal" urine. It is certainly wisdom in the beginner to adopt this axiom, and to act upon it in the routine examination and description of his patients.

(117) The disposition of "renal" œdema is determined in the first place by looseness of the cellular tissues, and in the second place by gravity; the disposition of "cardiac" œdema is determined in the first place by gravity, and in the second place by looseness of the cellular tissues. "Renal" œdema is first apparent, or is more marked, in the early morning; "cardiac" œdema is first apparent, or is more marked, in the evening.

(118) It is quite common to find concurrence of chronic nephritis and dilatation of the heart. Even so, it is important to determine which factor is the more dominant one in the case, because, if such determination is possible, the appropriate treatment can be more readily undertaken.

(119) Both chronic nephritis and dilatation of the heart may lead to a scanty, albuminous urine; but in the case of chronic nephritis the scanty urine is usually pale—pigment elimination being diminished

—is free from, or has but a small, deposit, the amount of albumen is considerable, and renal casts are present. Whereas in dilatation of the heart the scanty urine is generally high coloured, contains a large uratic deposit, the amount of albumen is usually low, and renal casts are absent.

(120) In nephritis, arterial tension rises for some time before arterio-sclerosis shows itself—a clinical observation that preceded the introduction of the sphygmomanometer by many years. Similarly, in nephritis the heart is affected before there are any signs of cardiac hypertrophy. This heart "irritability" shows itself by shortness of breath, palpitation, præcordial distress and night starts on the subjective side, and by arrhythmia on the objective side. These are functional disturbances, and they subside before any structural changes take place if renal adequacy is restored within reasonable time.

(121) It is a common practice to attempt to "flush the kidneys" in certain cases of nephritis in which the secretion of urine is diminished; but experience does not justify this measure; nor does theory, for in acute nephritis the use of copious drinks contravenes the main indication, which is to secure physiological rest of the inflamed organ, renal elimination of water being a vital, and not a mechanical, process; whilst in chronic nephritis such treatment tends to increase existing œdema, or to induce it if not already present. *The time to increase the fluid ingested is when diuresis begins, not before it has commenced.*

(122) The French have a maxim by which they express the urgent necessity of strict adherence to a diet of milk in any case of nephritis in which there is the least suggestion of acute uræmia: *lait*

au mort. The fear of starvation is so deeply rooted in the English mind that it sometimes becomes necessary, in order to secure co-operation, to meet this fear by arousing another of a worse contingency. Even when the decision to confine the diet to milk is agreed upon, the whole question is not settled; the matter of quantity is equally important; 30 oz. *per diem* should be the limit in an adult, and half that quantity in a child. The total amount of fluid allowed should not exceed twice these amounts (*vide* 121).

(123) The use of saltless diet in cases of renal œdema, though it has not justified the enthusiasm with which French physicians originally advocated it, is yet a helpful adjunct in treatment.

(124) Decapsulation of the kidneys in the treatment of nephritis has fallen almost entirely, but probably quite undeservedly, into disrepute. Like many another therapeutic measure it was killed by its friends, who advocated it without sufficient forbearance. Its success turns upon the careful choice of suitable cases. Given a case of sub-acute nephritis, or a case of acute nephritis which is tending to become chronic, in which dropsy is persistent, in which structural cardio-vascular changes are absent or are present only in slight degree, and in which thorough treatment on general lines has failed to establish a cure in three months—in such a case the operation should be seriously entertained. The mortality of the operation in such a case is very slight, and the results more than justify the procedure.

(125) The known presence of a calculus in the kidney is not in itself an indication for surgical treatment; but if, in spite of general measures, (i) chronic pain or attacks of colic persist, (ii) hæma-

turia recurs, or (iii) the urine becomes infected, operation should be advised.

(126) Tuberculosis of the kidney most often begins in the apex of a pyramid. This accounts for the frequency with which hæmaturia is the first sign, or one of the earliest signs, of the disease. The hæmaturia often intermits, and the urine may be quite healthy between the attacks. The hæmorrhage may be induced by exercise and may be checked by rest. The analogy with calculus in these respects is sometimes very striking, and, occasionally, may be rendered even closer by the occurrence in renal tuberculosis of pain of a colicky nature.

(127) The key to many intractable cases of *B. coli* infection of the urinary tract is often to be found in the state of the bowel.

(128) The occurrence of epididymitis as the result of B. coli infection is not widely known; the affection is therefore often attributed erroneously to gonorrhœa or to tuberculosis.

(*To be continued.*)

A New Pylorus.

POSTERIOR GASTRO-JEJUNOSTOMY WITH JEJUNO-JEJUNOSTOMY AS A ROUTINE OPERATION IN ALL CASES OF PYLORIC OBSTRUCTION—DUODENAL OR PYLORIC OR CHRONIC GASTRIC ULCER.

By G. GORE GILLON, F.R.C.S.
Late Lieutenant-Colonel attached R.A.M.C., and Surgeon, London Military Hospitals.

MY reason for writing this article in THE PRACTITIONER is to bring before the medical profession of this country a new and, I venture to say, an improved method of dealing with cases of gastric and duodenal ulcers, and of pyloric obstruction.

It appears to me that, in reviewing the results of the ordinary " no loop " operation for these diseases, surgeons are too apt to refer to their large number of such cases after operation as " quite well." What is meant, I take it, is that so far as the original ulcer is concerned they are " cured," but, as a large majority of general practitioners must admit, such cases have anything but a pleasant after-life to face, and the exception is to come across men who are " quite " well, able to do hard work, and digest ordinary food without the least discomfort.

In the past few weeks I have collected eight cases, in which the patients suffer great discomfort in their stomachs and have occasional vomiting.

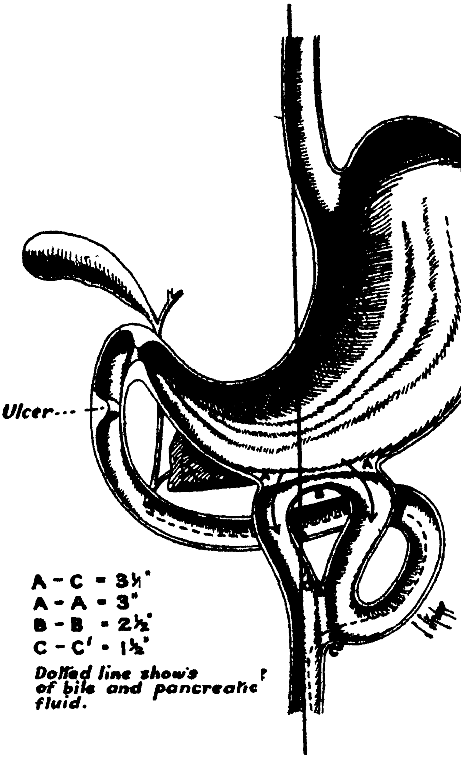
The obvious remedy for this state of things is to have an operation done, which produces a condition of an exact copy of Nature's own method of dealing with the upper zone of alimentary digestion, and I claim that the operation of posterior gastro-jejunostomy, with an intestinal anastomosis done at the same time, is the ideal procedure.

HISTORY OF THE OPERATION.

The history of this operation may be stated briefly. Braun, Weir, and Jaboulay performed jejuno-jejunostomy over 20 years ago, but *only* when combined with an *anterior* gastro-jejunostomy.[1] Jaboulay published his paper in 1894, and Weir in 1898.[2] Jejuno-jejunostomy was done occasionally, as Sir Berkeley Moynihan has informed me, after these dates in England, as an addition to the posterior operation when a very long loop had been left. But this method quite went out of fashion, for there were alleged against its use: (1) the occasional occurrence of a jejunal ulcer afterwards, and (2) the length of time involved in its performance. The "long loop" operation in the early days probably failed either because the loop was too long and so got kinked, or because the technique at the time was faulty and a certain number of cases of jejunal ulcer were reported as having followed as a sequel. The time for the double operation should never exceed 1 hour 20 minutes nowadays, allowing a few minutes for change of gloves for the second anastomosis. Thus this long loop operation was practically discarded 15 years ago, and surgeons all over the world have done the present operation of "no loop" posterior gastro-jejunostomy. This operation was and is undoubtedly a "cure" for cases of pyloric obstruction and duodenal and pyloric ulcer, for certainly the ulcer does heal and the obstruction is overcome; but there was much dissatisfaction as regards the results of the operation as time went on, many patients complaining of various uncomfortable sensations in the abdomen, and very many of them being in feeble health and unable to take hard exercise.

A NEW PYLORUS.

When one sets about altering the mechanism of the alimentary tract—altering Nature's plan—it surely behoves him to do so in a manner that shall produce no secondary disability. We must remember that the patient has presumably a long time to live with the modifications which the surgeon has effected. When we want to do away with the use of the old pyloric gateway we must make certain that the new gateway is situated at the *lower* end of the stomach (*see* figure), and by a jejuno-jejunostomy, some 3½ in. away from the new pylorus, we can



It will be observed that in this diagram of the completed operation there is no sagging of the loop left and the course of the bile, etc. is "downhill" to the P. and C′.

make sure that the food stream does not mingle with the bile and pancreatic fluids till the proper time. By a jejuno-jejunostomy the duodenum gets a perpetual rest, and the descending jejunal double tube takes the functional place of the first segment of the duodenum.

The accompanying figure shows my modified operation for short-circuiting the gastric contents.

I hold that the present-day "no loop" operation is physiologically incomplete; the bile and pancreatic fluids find their way into the stomach, and the long series of complaints noted by physicians are entirely and wholly due to this unnatural result of the conventional operation. These 3½ in. of jejunum shown in the figure functioning as the first 3½ in. of duodenum make all the difference in the world to the unfortunate sufferer. Digestion goes on naturally; he is never troubled with hyperacidity, and jejunal ulcers simply do not occur.

THE OPERATION DESCRIBED.

To prevent misconception, I will describe shortly my operation. After opening the abdomen and exposing the part of the stomach I require, I put in my right hand and bring up from the left of the spine 10 in. of jejunum, counting from the duodeno-jejunal junction, immediately at the left of the second lumbar vertebra; 3 in. of this proximal loop are used to make the first anastomosis, leaving 7 in. for the second anastomosis. The gastro-jejunostomy opening is made 3 in. in length (*see* A to A in figure). My assistant and I now change our gloves and then make the anastomosis between the two descending legs of the jejunum at a distance of 3½ in. below the stomach opening (B to B in figure). The anastomotic opening itself should measure 1½ in. vertically (C to C^1). The gastro-colic omentum is attached to the jejunum near the stomach by two stitches, and any veins in the omentum tied. I use fine silk or linen thread. The abdomen is then sewn up in the usual way. The vertical black line in the figure shows the position of the spine. A to A indicates the new pylorus, the arrows point out the direction of the food stream. The dotted line along the duodenum from Vater's ampulla to the jejunal opening, C to C^1, shows the course of the secretions from the pancreas and liver to join the food stream issuing from the stomach.

The patient can lie in a recumbent position a few hours after the operation, and need not be propped up; hence, there is less strain on the abdominal stitches. He is fed with tablespoonfuls of water for a day, then peptonized milk in the usual way for a few days, and in eight days is taking a fair amount of light food. On the twelfth day he gets from 2 to 3 gr. of calomel; prior to that his bowels are opened by rectal injections, if required. He should lie on his back for the first three weeks, and leave hospital on the twenty-fourth or twenty-eighth day. The operation has never taken me longer than 1 hour 15 minutes in an uncomplicated case. I have never had a case of jejunal ulcer afterwards.

RESULTS.

The results are uniformly good, and the patients are not only well but very well. They put on weight. They develop a great capacity for swallowing large quantities of liquids without discomfort. I may mention that three of my cases—two men and one woman—undertook a journey of some 1,200 miles by sea within a few months after the operation, and although all three had previously been great sufferers from sea-sickness, yet they reported to me afterwards that they were not seasick, and enjoyed their meals heartily all through the voyage, although it was winter time and the sea was very rough.

NOTES OF A CASE SHOWING PROFOUND ANÆMIA.

Mr. —, aged 30, height 5 ft. 10 in., was operated on by me at St. Thomas's Nursing Home on December 1, 1918. He was admitted to the hospital on November 16, 1918, suffering from profound anæmia due to loss of blood for the past twelve years from a duodenal ulcer. He was so pale and bloodless that he had to be kept fifteen days on special diet and have intravenous saline injections before he was fit for operation. His weight then was 8½ stone. The operation, in which Mr. O. H. Hyman assisted me, took exactly 1 hour and 15 minutes. Three weeks after the operation the patient left the hospital, and in six months' time he put on over 1½ stone in weight. He went back to work in April, 1919, and has been at work ever since, and has not the slightest sign of indigestion or any pain or inconvenience of any kind; his cheeks are a healthy red colour.

With regard to my experience of this operation, I may say that during the last nine years I have carried it out in the way I have described in every case. As a routine practice so convinced am I of its excellence that I adopt it in cases of perforation, either at the same sitting, or, when the patient was too weak, by opening the abdomen again in four weeks' time, and leaving meanwhile a good 7-inch loop of proximal jejunum for me to find when I make my second anastomosis.

I should be much obliged if medical men would send me a list of cases in which the after-results of the "no loop" operation have been unsatisfactory, giving the date of the operation.

REFERENCES.

[1] Vide *Archiv. f. klinische Chir.*, 1823, xlv., pp. 3614 (Braun).
[2] *Med. Record,* 1898, vol. liii., p. 541.

The Surgery of Gastric Urgencies.

By RICHARD WARREN, M.D., M.Ch., F.R.C.S.
Surgeon to the London Hospital; Surgeon to Brompton Chest Hospital, etc.

IN this article I shall refer only to two classes of gastric urgency, viz., (1) perforation of, and (2) hæmorrhage from gastric and duodenal ulcers. Other urgent conditions form a very small part of the cases encountered in civil practice. Wounds of the stomach are very like wounds of other hollow viscera, and are amenable to the same treatment. Foreign bodies in the stomach, which will not pass the pylorus, are easy enough to remove by simple gastrostomy, and it is only impaction in the cardiac orifice that is likely to lead to surgical difficulties; in which case, we can usually extract them by the aid of the œsophagoscope. The results of swallowing corrosive fluids are seldom referred to the surgeon, in the recent stages; and rightly, for if enough has been swallowed to produce perforation and peritonitis, there will be too much destruction of the whole stomach to give any prospect of surgical measures being successful. In the later stages, when the amount swallowed has been less and there is stenosis consecutive to ulceration at the pyloric and cardiac orifice, surgery will be of the greatest assistance, but such cases can hardly be considered urgent.

PERFORATION OF GASTRIC AND DUODENAL ULCERS.

In this series are 72 cases on which I have operated between the years 1910 and 1919. Perforation of an ulcer may occur at practically any point in the stomach down as far as about the end of the first part of the duodenum, showing the importance of the alkaline efflux of the biliary and pancreatic ducts in preventing such ulceration. Below the first part of the duodenum perforation is very uncommon, though I have seen two cases of perforation of ulcers in the upper jejunum. The site of election for perforation of peptic ulcers is just on the duodenal side of the pylorus. Of 72 cases, 25 were definitely gastric, 37 definitely duodenal, and 10 were in the pyloric region but of doubtful situation. When there is a good deal of lymph lying around the perforation and the patient is very ill, one cannot spend much time in exactly localizing the position of the perforation with regard to the pyloric vein.

Mortality.—This may appear high, but it must be considered that there has been no selection of cases, they run in a consecutive series and operative treatment was never refused unless the patient was in articulo mortis. Of 25 gastric ulcers, 10 died; of 37 duodenal ulcers, 12 died; and of 10 cases, in which the situation was not exactly determined, 7 died; this last group includes the worst cases. In other words, one-third of the ulcers were in the body of the stomach, the remaining two-thirds in the region of the pylorus and duodenum. The pure gastrics work out to a mortality of 40 per cent., the pure duodenals to one of 33 per cent., but if the cases of uncertain position in the vicinity of the pylorus are included with the latter, the pyloro-duodenal group has a mortality practically the same as the gastric. An important point in prognosis is the length of time an ulcer has perforated before operation. Cases of over 24 hours' duration have a very bad prognosis; nevertheless, we have known cases perforated two to three days occasionally recover, while sometimes those caught very early, inside 12 hours, will succumb. There can be little doubt that the contents of the stomach vary greatly in effective virulence. As in other forms of acute peritonitis, the prognosis can better be made from the general appearance of the patient as regards face and pulse than from the appearances of the interior of the peritoneal cavity. The mortality, I am glad to say, shows some improvement as the series proceeds. The total mortality is 40 per cent., but while of the first 30 cases it is 47 per cent., for the last 30 it is reduced to 27 per cent., or almost half. Various methods have been employed in dealing with these cases, and it is with the idea of finding out the relative merits of these modifications that this series has been investigated.

Mere practice and consequent increase in skill probably accounts for some improvement. Thus in the early part of the series the average duration of the operation, including both cases in which gastrojejunostomy was done and those in which suture of the perforation only was performed, was 50 minutes, while in the latter part of the series the average duration of the operation in cases which included gastro-enterostomy was 29 minutes. The reduced handling of the abdomen indicated by the shorter operation is favourable to recovery.

The variety of ulcer in the two series was as follows:—

	Gastric.	Duodenal.	Doubtful pyloric.	Recovered.
1st 30 - -	12	16	2	53 per cent.
2nd 30 -	7	17	6	73 „

which suggests that the second series was at least as severe as the first.

One of the points somewhat under contention in the treatment of these cases is whether gastr jejunostomy should be performed as part of the emergency operation or not, the following figures are to hand :—

1st 30 gastro-jejunostomies - - - -	21
2nd 30 „ - - - -	25

In the series with more gastro-enterostomies the mortality was lower.

Methods of Treatment adopted. — The method employed varied according to the condition of the patient, in most instances the ulcer was occluded with one or more sutures, then invaginated with a second row, after which posterior gastro-enterostomy was performed and the pelvis drained for 48 hours. In two cases following simple suture of the ulcer, a gastro-jejunostomy was done after a few days. In a fair number the patient was not considered fit enough for a gastro-jejunostomy and, especially if the ulcer was in the stomach, simple suture in layers was done. Not satisfied with the results from this method, and thinking that malnutrition from insufficient feeding played an important part in lowering resistance and diminishing power of healing, a jejunostomy for feeding was done as well as suture of the perforation in layers. In other cases, after closing the perforation with one or two sutures, a gauze pack was placed down to the perforation and the pelvis drained. The results were as follows :—

Suture only.—Lived, 6; died, 9. Total, 15. Recovery rate, 40 per cent.

Suture and immediate gastro-enterostomy.—Lived, 34; died, 13. Total, 47. Recovery rate, 72 per cent.

Suture and gastro-enterostomy after a few days.—Lived, 1; died, 1. Recovery rate, 50 per cent.

Suture and jejunostomy.—Lived, 1; died, 5. Recovery rate, 17 per cent.

Pack and drainage.—Lived, 1; died, 1. Recovery rate, 50 per cent.

The line taken was to perform gastro-enterostomy wherever possible, especially if the ulcer was in the duodenum or pyloric zone of the stomach, and the patient seemed to stand a reasonable chance of enduring a few more minutes operating.

Late Results.—Attempts were made to follow up these patients; of the 43 survivors information was obtained about 14, the time since operation varying from three to ten years. All the patients were in good condition without return of symptoms, and in all except one gastro-enterostomy had been done. One patient with gastro-enterostomy died of intestinal obstruction a few months after the original operation, as did also one of the cases in which suture only had been done. I am inclined to think that the addition of a gastro-jejunostomy improves the patient's chances in most instances by diminishing the acidity of the gastric contents and by enabling us to feed the patient earlier. One has often been struck that these patients are suffering from malnutrition due to insufficient feeding and for that reason will not heal well. The addition of a gastro-jejunostomy adds but little to the duration of the operation when performed by one who is frequently operating on these cases, as has been pointed out above. On the other hand, as such cases are very urgent, it may happen that it is necessary for a surgeon of small abdominal experience to operate, when it will be wiser to rest content with the least complicated procedure, and not undertake a more perfect operation which may prove difficult and perhaps take an hour or more to finish. For such cases when the operative experience is small or the conditions under which operation has to be done are unsatisfactory, it is advisable simply to occlude the perforation with one or two sutures taking a wide grasp of the stomach wall around, placing a gauze pack down to this, swabbing out the bulk of the effusion in the vicinity of the stomach and especially between the liver and diaphragm and in the right kidney pouch and pelvis. The operation is concluded by closing the upper part of the wound except for the gauze pack and draining the recto-vesical pouch above the pubes with a wide tube for 48 hours. The plan of performing a jejunostomy was adopted in very bad cases with the idea of getting nourishment more readily into these very debilitated patients. The results do not appear good, nevertheless most of these patients showed considerable improvement for a while, but as only the more hopeless cases were treated in this manner the mortality was naturally severe. In fact, when the patient is unfit to stand a gastro-enterostomy, performed with expedition, it will be very difficult to save him by any method.

HÆMORRHAGE FROM GASTRIC AND DUODENAL ULCERS.

This condition, as one for operation, is much less common than perforation, only 12 cases having come under my care. The disparity is probably hardly so great in reality, but hæmatemesis is a less obviously urgent sign than perforation, and is more likely to be treated expectantly by physicians, often with disastrous results. For bleeding from gastric and duodenal ulcers is a variety of secondary hæmorrhage, and, as such, demands operative interference urgently. This delay accounted for some of the deaths in this series. Of 12 cases, 5 died; 58 per cent. recovered; but of these five one died on the table and two others shortly after, being too reduced by prolonged bleeding to stand even 20 minutes operating; another case which died was one of acute duodenal ulcer following on an amputation of the breast done by another surgeon, which had suppurated severely and was practically a case of septicæmia. If the surgeon was allowed to operate earlier in these cases, the mortality would be quite low. Ten cases were gastric, two only being duodenal. The ulcer was usually on the lesser curvature, eroding branches of the coronary and pyloric anastomosis. In one case only, that of a young girl, there was no definite indurated ulcer, but simply congested swollen mucosa dripping blood from tiny ulcers, the condition sometimes described as gastro-staxis. The technique employed was, after exposure of the stomach to pass sutures around the vessels of the lesser curve at each end of the ulcer and to cut off the blood supply at its source and then pass three or four stout catgut sutures, encircling the whole ulcer transversely to its long axis, the sutures passing into the cavity of the stomach. These were drawn tight and effectively crushed up the ulcer and prevented any possibility of bleeding, a posterior gastro-enterostomy concluded the operation. The lesson to be learned from this series is the importance of not delaying to operate in cases of severe hæmatemesis when the history points to a chronic gastric ulcer; the idea of waiting 24 hours to let the patient recover, or till there is another attack of bleeding is quite against all surgical principles. If the case is one of chronic ulcer with a fair-sized vessel eroded in the floor of the ulcer, there is nothing to be gained by waiting, all the probabilities are in favour of more bleeding taking place and the patient's condition becoming worse. Consequently, we consider severe hæmatemesis as an indication for immediate operation in cases in which there is reasonable expectation of finding a chronic gastric ulcer; when the condition is one of so-called gastrostaxis, usually in young girls, the indications are less urgent, for these cases seldom die, but as a gastro-enterostomy appears to clear up the condition, and as chronic ulcers are not unknown in young persons, we should not be inclined to wait very long in such cases before advocating laparotomy for severe hæmatemesis.

The Wassermann Reaction.

By J. STAVELY DICK, M.B.

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THE Wassermann reaction is a product of the twentieth century. Only a few extremists—friends or foes—would venture so early to define its level of usefulness with precision or finality. Mr. McDonagh, however, in the January, 1920 number of THE PRACTITIONER, makes an attempt to do so, and it is mainly in reply to his criticism that I offer some remarks.

My claim to be heard as a witness on this subject is based on the consideration that I have done over five thousand Wassermann tests by various methods since 1910, and that I have (with a dozen or so exceptions) seen all the patients personally. I seriously question the wisdom of any system which results in pathologists and clinicians working in watertight compartments; the more closely they are in touch with each other and with the patient, the less likely is there to be a weak link in the chain of evidence or any serious conflict of opinion. During the first few years of this period I worked almost exclusively with Fleming's simplified method, as I had seen it carried out at St. Mary's. The results left a good deal to be desired, and, if my experience was confined to this method alone, it is doubtful whether I could get up sufficient enthusiasm to record my impressions. I can, therefore, appreciate and sympathize with the adverse criticisms of Wassermann results, which one sees from time to time. I think the Local Government Board were extremely well advised when they ruled out of their scheme this and other "simplified" methods. Even a Government Department should get credit, where credit is deserved. Their decision has done much to evolve order out of chaos.

During the last few years I have done the test by Professor Dean's method, in which falling quantities (1–5; 1–10; 1–20; 1–40) of the patient's serum are tested with three minimum hæmolytic doses of complement, which is titrated immediately before use. I have more recently adapted this to Harrison's drop method, which saves time and diminishes the risk of double dosing a row of tubes when comparatively large numbers are being dealt with. I must confess I am enthusiastic about the test as here outlined—a somewhat rare experience after ten years' work. I feel that the Wassermann test is the most important and most reliable single test in routine clinical pathology. It is perfectly clear to me that this, or possibly some simpler method of ascertaining the extraordinary change which constantly occurs in the blood in response to syphilitic infection, has come to stay, even if it should cause annoyance on occasion to the patient or his doctor, as Mr. McDonagh suggests.

Assuming, then, that the test is done in such a way as to comply with the Local Government Board's requirements, that no technical error of any kind has occurred, and a positive result is obtained, what does it mean? It means, I think, in this country that the patient has contracted syphilis. But it cannot be emphasized too strongly that this test, in common with all others of the kind, is merely one item of evidence. It happens only rarely that a diagnosis is properly based upon one item of evidence alone. Everyone who has considered the subject carefully will agree, I think, that the most careful clinical examination is imperative always. I do not think that pathologists can fairly and justly be blamed because some clinicians rely too much perhaps on pathological evidence and too little on facts elicited by themselves. A wide survey of the whole field, and cordial co-operation between the two classes of workers are required in order to secure the best results. But it happens from time to time that a positive Wassermann is the only available evidence of syphilitic infection, or that the clinical features are so ambiguous as to be useless for the purpose of practical diagnosis. I may quote an illustration or two.

A patient was sent to me some five years ago in order that he might have an intravenous injection of salvarsan. He suffered from slight neuralgia. The history he gave was interesting. A year or two before I saw him he had suffered from persistent and apparently excruciating trigeminal neuralgia. He had consulted quite a number of specialists and ultimately his Gasserian ganglion was excised, but with only two or three weeks' relief from pain. It then occurred to someone to have the question of syphilis gone into; his blood gave a positive reaction. He was treated with salvarsan; his pain almost immediately ceased

This patient has had in the last five years three or four more intravenous injections, because he insists upon having one if he feels the slightest twinge of pain. But for all practical purposes the first intravenous, which he had before I saw him, cured him of his neuralgia. He remains well up to date, though his blood reaction is still strongly positive. In this case, as the neuralgia was the only obvious symptom, the failure to make a correct clinical diagnosis was almost inevitable.

A British soldier developed a sore throat about the middle of December, 1918, in Flanders. He was under treatment in billets until January 11, 1919; he was then sent into hospital. The first diagnosis on his case-sheet in hospital was diphtheria; swabs were taken and were negative. The diagnosis was changed to tonsillitis, and later on this was altered to septic throat on his case-sheet. He was demobilized out of hospital on March 16, and I saw him about a week later. The clinical features did not suggest syphilis; but, having regard to the history extending over three months, I tested his blood and was somewhat surprised to find it strongly positive. His throat cleared up with the usual rapidity after treatment with novarsenobillon.

This man had been under at least five (he thinks six) medical officers, and, as I have stated, I did not seriously consider that his throat trouble was syphilitic upon the clinical evidence. It is very likely that there are clinicians with a wide experience of cases of the kind, who would have made a correct diagnosis upon the clinical evidence alone. But such super-men are not always, or often, available for the solution of routine problems of the kind. The practical value, therefore, of a positive Wassermann is frequently decisive in diagnosis.

But if a negative result is obtained, what does it mean? Mr. McDonagh says it "means nothing." Some months ago a patient with a scrotal lesion was sent up to me from the clinical department of the V.D. Clinic with his card marked "tertiary syphilis." The Wassermann test was negative. Enquiry later elicited the fact that it had proved to be malignant.

Another example may be cited.

A patient came up at the same clinic with his card marked "secondary syphilis." He had a profuse eruption on the face. The Wassermann test was negative. A histological examination revealed the fact that the rash was tuberculous.

I may say that in both cases I myself was quite in agreement with my colleagues from the clinical standpoint. We all felt, however, I think, that in these instances a negative Wassermann meant *something.*

It is quite true that one fails in a considerable percentage of chancres to demonstrate the *spirochæta pallida.* One fails because it is really a soft chancre, or owing to previous local treatment, or owing to insufficient time being given to the search. Whatever be the cause of failure, a negative result is here very unsatisfactory. In this case a negative result may perhaps be said to "mean nothing." But in such cases, and they are by no means uncommon, I suggest that a negative Wassermann once a week or fortnight for a few weeks *means a great deal.* Indeed, I think that it may, with perfect safety, be laid down that a negative Wassermann in the presence of an active, untreated, and apparently syphilitic lesion is extremely important evidence. In this connection, I should lay stress on the word "untreated." I may cite a case in point.

I saw a private patient with an active throat lesion which her doctor thought was syphilitic. In this view I fully concurred. The Wassermann test was negative. Repeated a week later, it was still negative; but it was positive a fortnight later, treatment having been discontinued in the meantime.

The doctor told me that this patient was extremely susceptible to mercury, salivation occurring with quite small doses. Whether patients who are unusually susceptible to the influence of mercury are more likely to have a negative Wassermann than average patients is a point of interest, which I am at present unable to decide. But while a negative Wassermann may in certain circumstances afford extremely important evidence, it is obvious that it is merely one link in a chain. It is not the chain; it is really most desirable that this elementary distinction should not be lost sight of. Provided the technique of the test is correct, a negative reaction always means that at the moment the patient's serum fails to show the characteristic which is practically pathognomic of syphilitic infection. If one makes the deduction that the patient has not got syphilis, the deduction may be incorrect. But to say that a negative reaction means nothing, merely because an incorrect deduction may be made from the fact, is a line of reasoning which would soon play havoc with all signs and symptoms in clinical medicine.

I may cite an instance to illustrate what I mean.

I had a patient some seven or eight years ago who was examined *per rectum* by a distinguished provincial surgeon and pronounced to be suffering from inoperable carcinoma of the prostate. An even more distinguished London surgeon, after a similar examination, expressed the opinion that my patient was suffering from carcinoma or tubercle of the prostate, but that it was operable. After operation the surgeon, on inspection of the prostate, held that it was tuberculous; but he, very kindly and very fairly, sent me a histological report a few weeks later which stated that there was no evidence of carcinoma or tubercle. It was inflammatory. The patient is now in perfect health. Palpation revealed that there was swelling and more or less hardness. Inspection revealed a number of necrotic areas with small localized abscesses. Palpation and inspection were not at fault at all; what was at fault was the deductions made from the ascertained facts.

I cannot, however, imagine that anyone would conclude generally that swelling, hardness, etc. "mean nothing," because, in this and probably many other instances, incorrect conclusions have been drawn from the facts. A Wassermann test is quite analogous to palpation and inspection; it is a means of finding whether a certain condition of blood-serum does or does not exist. The presence or absence of this serum change inevitably means something. What it means exactly in any individual case is for the trained observer to say, after he has fitted in each link in the chain of evidence.

With regard to this reaction as a guide to treatment and prognosis, Mr. McDonagh says "more good can be done by telling a patient that it is useless, than by prescribing an unlimited number of courses of arsenobenzene." It is difficult to deal with such a statement seriously. But I suggest that two things are clear—

(1) It is the business of the physician to advise upon treatment to the best of his ability.

(2) The propriety of telling a patient something as a fact, which is still *sub judice*, is open

to question.

Up to the last dozen years or so syphilitic patients were generally advised to continue under treatment for two years at least after the disappearance of symptoms. Under this *régime* some were cured, and some were not cured. It was not possible for physicians, however, to say in which category any individual patient should be placed.

To-day we are able to divide syphilitic patients, whose symptoms have cleared up, into two classes—

(1) those whose blood becomes negative under treatment, and remains negative after treatment is discontinued;

(2) those whose blood remains positive in spite of treatment.

This classification marks an advance of extraordinary interest. But is this merely an academic or is it a practical classification? In other words, does a permanently negative serum reaction mean cure, and a persistently positive reaction mean failure to cure and liability to late sequelæ? If so, will intermittent treatment be of any avail in postponing or averting the danger which threatens?

Some time ago I saw a patient aged seventy-six. He suffered from early locomotor ataxia. His serum gave a positive reaction, and he admitted that he had syphilis when about twenty; that is to say, fifty years or more prior to the onset of tabetic symptoms. It need not take fifty years, however, for the solution of these problems. What is required for their elucidation is careful records, which are now being accumulated; keenness in following up the future history of patients at present under treatment and classification; and, above all, a reasonable term of years during which we shall refrain from formulating conclusions, which are not based upon well-ascertained facts.

A Recent Analysis of Consecutive Induction of Artificial Pneumothorax in 50 Cases of Pulmonary Tuberculosis.

By Z. P. FERNANDEZ., B.A., M.D.

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THE value of artificial pneumothorax in suitable pulmonary tuberculosis has of late been so well recognized by the majority of tuberculosis physicians that the subject cannot but be of interest to the general practitioner in selecting such cases. It is an irony of fate that this treatment, which originated in this country, should have been ignored till of late, only to be widely adopted abroad.[1] As early as 1821 A.D., James Carson of Liverpool experimented on rabbits and prophesied a future therapeutic value before the local Medical Society. The Clinical Society, London, applauded William Cayley for the first case of hæmoptysis arrested by induction, though with a technique crude beyond doubt. Soon Potain in France, Forlanini in Italy, Murphy in the States, Brauer in Germany, L. Spengler in Switzerland, Saugman and Hansen in Denmark, and a number of other foreign clinicians took up the treatment with success. In England, the method was reintroduced by Dr. Claude Lillingston.[2] A personal account of his case, appearing in the Special Tuberculosis Number of THE PRACTITIONER (January, 1913), is worthy of note. A patient at Mesnalien Sanatorium, Norway, with febrile pulmonary tuberculosis in 1909, he took the risk of the treatment at the suggestion of Dr. Holmboe, with the result that his temperature became normal, and his cough and expectoration ceased. The next year he was able to return to England, and, with Vere Pearson, performed the first induction at Mundesley Sanatorium on a patient who was alive two years later. The first publications on the subject appeared[3] in July, 1911, by Lillingston, Vere Pearson, Colebrook, and Rhodes.

Space forbids the analysis of the various Continental results so far published; but a study of the literature on the subject confirms my opinion that there is considerable beneficial effect in 40 to 50 per cent. of cases successfully treated, in spite of the advanced material selected, with general arrest of severe hæmoptysis. The results may still be improved with a slowly acquired clinical instinct to select the suitable case, or with X-ray or fluoroscopic examination. Dosage and timing of interval, too, have in the past been haphazard. The figures of Sachs (1915)[4] on 1,145 cases of 24 American observers with about 50 per cent. benefit, and of Greer (1918)[5] on 32 cases of 3½ years' duration with 44 per cent. benefit, compare favourably with the satisfactory result detailed below.

Roughly estimated for a period of 18 months from September, 1917, to March, 1919, about 800 pulmonary tuberculosis patients of all stages were treated at the Leeds City Seacroft Sanatorium. Keller, according to Riviere[6] (THE PRACTITIONER, 1914), estimated 7 per cent. as suitable. Considering intermediate and advanced cases, about 10 per cent. have been subjected to induction treatment. Of the 40 per cent. early cases with few physical signs and systemic disturbance, the majority improved under sanatorium conditions; only seven of these, who showed progressive activity, were selected for the treatment. The average percentage mortality for six years in the sanatorium was about 16·7 of all discharges. In the official year ending March, 1918, the life-time of the patients who died in the sanatorium averaged one year and five months after the first definite symptomatic manifestations, as against two years and one month of those who died with previous induction treatment. The majority of the treated cases still alive after two years had then a period of illness dating on the average two years and four months. The patients treated were of the working class, the majority of whom did not seek advice until the symptoms were alarming and signs pronounced.

Severe and recurrent hæmoptysis is a frequent and occasional fatal symptom in pulmonary tuberculosis of the above type of patients. Exacerbation with fatal termination often follows such hæmorrhage. In 1917 to 1918, I witnessed nine sudden deaths from hæmoptysis, about 6·6 per cent. of the total mortality. For six years, about 1 per cent. of all admissions and 8·6 per cent. of all mortality died of this complication. In such cases when medical treatment fails, so long as there is no cardiac or renal disease, induction can be more safely relied on than surgical interference for an abdominal catastrophe. The anxiety of friends and the responsibility of the physician are thereby greatly relieved. Fifteen such cases have been classified below, including eight published in July, 1918,[7] on "The Value of Artificial Pneumothorax in the Arrest and Prevention of Hæmoptysis in Pulmonary Tuberculosis." Another baffling symptom is continued high febrility, from which 55 per cent. of the fatal cases suffered. In 15 out of 18, febrility was arrested by induction in less time than by the usual sanatorium measures.

The apparatus I have found of great value is that of Dr. H. de Carle Woodcock, the visiting physician. Unless the working is well understood and the tubes and glasses kept scrupulously clean from dust, the novice may truly fail in his attempts. It is worth while to test the apparatus before an unnecessary puncture is made. Leeds Sanatoria rank among the pioneer centres in England where the treatment was introduced by Dr. Woodcock. To him, and to my predecessors Dr. J. A. M. Clark, Dr. N. Gebbie, and Dr. J. J. Jervis, I am greatly indebted for including a few cases directly treated by them, which came to my care subsequently in the period under consideration. To estimate the time-factor regarding benefit derived, these cases have been of great value.

Result in March, 1920 *of the* 50 *Cases treated.*

GROUP I.—*Fifteen cases of severe and recurrent hæmoptysis* (*six unilateral and nine bilateral*). Death in five.

(*a*) One unilateral case after temporary improvements of one year and four months died of bilateral disease;

(*b*) Of the four bilateral, one died of influenza in the epidemic of November, 1918, and three treatments not continued.

Ten are now alive, of whom eight show quiescent signs. Four of these are fit for a full day's work, one for over 2½ years, two for over two years and four months, and another for 1½ years. Four are fit for light work; two for 2½ years and two for two years and three months. The remaining two have done light work for over nine months, and the condition of the lung is one of chronic fibrous disease. Hæmoptysis has been successfully arrested in all but one, who refused the treatment for ten months in spite of recurrence and severity. The day after he consented for an initial induction of only 200 cc. of oxygen, he had a severe fatal hæmoptysis. Necropsy revealed a cavity at the right base of the lung, and the pleura was about half-inch further on the right side than on the left. My impression was that the patient would have derived benefit with earlier and continued induction treatment, and that the hæmoptysis was independent of the induction, the quantity of gas used being too small.

GROUP II.—*Nine cases of unilateral softening of more than one lobe with moderate resistances and systemic disturbances.* Death in two after temporary improvements of twelve and eighteen months respectively. Alive, seven with quiescent signs and satisfactory working capacity, including two cases of six and four years after first induction.

GROUP III.—*Five bilateral cases. Extensive disintegration of one side and moderate infiltration of the other. Resistance good and systemic disturbances tolerable.* Death in two, one two years after from influenza and another six years after the first induction. In the three alive, the lung condition is quiescent and capacity for work good after 3½, 4½ and 5½ years respectively.

GROUP IV.—*Thirteen bilateral cases as above type but resistance and systemic disturbances not satisfactory.* Eleven of these are now dead. Five from influenza epidemics with considerable improvement previously. In six treatment could not be continued owing to adhesions or other complications. In two now alive condition satisfactory after 1½ and 2½ years respectively.

GROUP V.—*Four bilateral advanced disease with slight negative pressure and severe systemic disturbance and poor resistance.* Death in three and treatment could not be continued owing to adhesions or other complication. No improvement in the fourth after 1½ years.

GROUP VI.—*Four cases of advanced bilateral disease with terminal laryngeal complication.* Death in two after two years, one after four years, and another nine months after first induction from laryngeal complication after considerable general improvement.

Briefly, in September, 1919, out of 50, 25 died, and in four the prognosis was bad, of whom three died since. In 21, or 42 per cent., of the cases treated the condition was satisfactory in March 1920. In seven death was directly from influenza, and in others that died the treatment was not continued, or they had died after considerable improvement. Of six patients who had bilateral induction, three are dead and three have done light work for 2½, 3½, and 5½ years respectively.

It is interesting to compare the above figures with an analysis of Vere Pearson in July, 1919[8] of 44 cases treated from 1910 to 1916, of whom 22 were alive, and 42·6°/₀ or 9 out of 21 of the effective induction cases were in good health. The patients were no doubt of a better social status and environment.

In the mode of induction of the above cases the writer experienced no dangers, and pleural effusion resulted only in one case. The gas used, except in my early experience, was invariably oxygen. Following precedence, I have used quantities from 1,200 cc. upwards only in a few cases. But my experience, also confirmed by that of Dr. Woodcock and other recent authorities, is that small doses of usually 300 to 500 cc., repeated at frequent intervals, not only avoid distress to the patient but achieve satisfactory results. Mediastinal and cardiac displacement never follow such procedure. The manometer is the best indication for gauging the doses, and the writer never aims to alter the negative pressure to one considerably positive. From my experience in in-patient and out-patient work of over 5,000 cases, the benefit of artificial pneumo-thorax is beyond question in selected cases. To those interested in the subject, the words of William Ewart,[9] seven years ago, are well worth quoting: "Forlanini's non-bacillary mechancial cure was a singular instance of a method, the offspring of a master thought, the full panoply and remedy for immediate action, but too heroic, too magnificent, for practical tactics, fraught with hunting risks greater than have yet been registered, and therefore bound to pass away as ineligible for universal cure, yet imperishable in its teachings, which I venture to believe contains that cure."

In conclusion, for the initial attention the cases received at the Central Tuberculosis Dispensary and for every help I have received, I express my appreciation to Dr. H. de Carle Woodcock. To Dr. A. E. Pearson, the Medical Superintendent, I am thankful for the facility and encouragement he has given me in carrying on the treatment.

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Diseases of the Liver and the Vicious Circle.

By JAMIESON B. HURRY, M.A., M.D.
Late President, Reading Pathological Society.

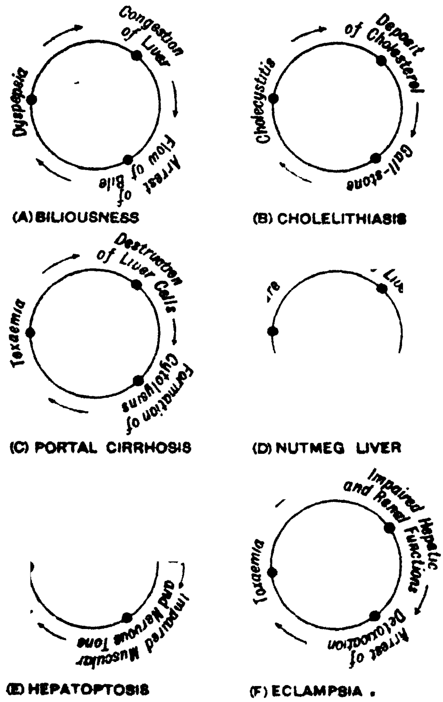
THE liver is the largest and most complex gland in the body, and discharges a great multiplicity of functions. Its metabolic and detoxifying activities are of fundamental importance, while the *rôle* played in digestion and excretion is indispensable to good health. So important an organ has, as might be expected, the closest reciprocal correlations with other organs both for weal and for woe. Lancereaux writes :—

> "The physiological solidarity which unites the various organs of the body is so intimate that one cannot be disturbed in its functions without the others being thrown into disorder. The liver is subject to this general law. On the one hand, its functions may be disturbed by disorders of other organs, while, on the other hand, hepatic disorders react injuriously on other organs."[1]

Physiological correlations present advantages as well as disadvantages. A great advantage is that they allow one organ to afford relief to other organs in distress. Thus the liver is able to render vicarious assistance to a disordered digestive, vascular, renal, or other system, and such assistance frequently enables the physiological self-regulating mechanisms to restore the disordered functions to their normal condition.

In the case of serious disease, however, physiological correlations are apt to become pathological correlations. The primary disorder provokes a secondary disorder, which then aggravates the first, and so the morbid process ***vires acquirit eundo.*** Vicarious assistance ends in a vicious circle.

Such injurious reciprocations possess great clinical importance, and exert a dominant influence in the mechanism of disease. In the case of the liver, they are too numerous to be adequately dealt with in a single article; but a few striking examples may be described. They may be conveniently discussed in connection with the digestive, the nervous, the vascular, and the renal systems.



(A) BILIOUSNESS (B) CHOLELITHIASIS

(C) PORTAL CIRRHOSIS (D) NUTMEG LIVER

(E) HEPATOPTOSIS (F) ECLAMPSIA.

The Vicious Circle in Hepatic Disorders.

I.—THE DIGESTIVE SYSTEM.

Various *circuli vitiosi* result from the close interdependence of the liver and the gastro-intestinal tract. Lauder Brunton thus describes the pernicious correlations:—

> "Indiscretion in eating or drinking disturbs the digestive processes in the stomach and intestines; the products of imperfect

> digestion or of decomposition in the intestine being absorbed into the veins pass to the liver; they may there induce an obstructed flow through the hepatic capillaries; the venous blood returning from the stomach and intestines will no longer be able to find an easy passage into the general circulation, and venous congestion of the stomach and intestines will be the result. Such venous engorgement as this will interfere with gastric and intestinal digestion, and this again will react upon the liver. Here, then, is a vicious circle which it is necessary to break."[2]

Biliousness.—A common example is afforded by the disorder popularly known as "biliousness." This frequently originates in some dietetic error which sets up gastric fermentation with the production of irritant lactic and butyric acids, followed by the secretion of an excess of mucus. The irritated stomach, no longer able to convert its contents into peptones and chyme, expels into the duodenum semi-digested materials which are entirely unfit for intestinal digestion and absorption, and only serve to disturb the physiological processes in the intestines. Meanwhile angry reflex messages are continually being dispatched to the liver and pancreas, perverting the functions of these glands and interfering with the due discharge of their secretions. The hepatic antitoxic functions are also largely arrested, so that organic poisons which reach the liver from the portal system continue in circulation and travel to the remote outposts of the economy. Thus the primary disorder spreads far and wide, with echoing reverberations from one organ to another. Bigg describes the process :—

> "Vitiation of the bile is a disorder of common occurrence, and is usually the result of absorption of deleterious substances which have been brought in the circulation to the liver. The most frequent cause is an unhealthy accumulation in the intestines, but the unhealthy accumulation most frequently is dependent on the vitiation of the bile, so that both conditions are dependent on one another for their causation. A vicious circle is established, and there is a simultaneous progressive increase of accumulation and of vitiated bile. The question of priority of causation may be an interesting scientific problem, but is not of any practical importance, since both conditions co-existing and acting as cause

> and effect, the cure of either condition must necessitate the removal of both conditions."[3]

Jaundice.—Another common hepatic disorder is jaundice, which is also perpetuated by a vicious circle. Catarrhal obstruction to the biliary flow causes bile-salts and bile-pigment to pass into the blood, where they may cause hæmolysis and liberate hæmoglobin. This leads to an excessive formation of bile-pigment, which in its turn gives rise to an increased viscidity, and to further obstruction of the flow of bile. Even a slight obstruction may entirely arrest the flow. Wells writes:—

> "Since the bile-salts cause hæmolysis, and since even in 'hæmatogenous' jaundice they may enter the blood, it can readily be seen that in this way an increased formation of blood-pigment may be incited which leads to further obstruction to the outflow of bile from the liver, and a vicious circle may thus be established."[4]

Hepatic Insufficiency.—Severe disorders of the digestive system may provoke the grave condition known as hepatism or hepatic insufficiency, which results when the liver fails to arrest poisons, which in their turn further depress its antitoxic activities. Monod describes the process:—

> "By hepatism we mean a diathesis, hereditary or acquired, characterized by inadequacy of the liver cells. A vicious circle is set up, the toxins set free reacting in turn on the cells of the liver."[5]

A similar but more virulent process operates in those forms of hepatic insufficiency associated with acute yellow atrophy of the liver and phosphorus poisoning, although the nature and exact mode of action of the poisons is not known. In the most typical cases the poisons exert a highly specific action on the liver cells, destroying their synthetic activities without injuring their intra-cellular proteolytic enzymes. Consequently, autolysis takes place, and the organ shrinks sometimes to a half of its former weight. The resulting loss of antitoxic activity allows the toxins to continue in circulation, cause and effect reacting on each other and resulting in a cumulative toxæmia.

In the worst cases there may be rapid self-digestion of the liver cells, giving rise to the violent toxic conditions of acute yellow atrophy.*

In many forms of hepatic insufficiency an injurious circular process appears to be connected with acidosis. If the fatty acids are not fully oxidized, free organic acids are left over which combine with ammonia and alkaline bases of the blood. Consequently, these bases are no longer available for preserving the alkalinity of the blood, which diminishes and gives rise to acidosis. This acidosis in its turn accelerates the breaking up of liver cells, since autolytic enzymes act best in an acid medium. Wallace and Gillespie write:—

> "When once the condition of acidosis has been established a vicious circle is formed, since the more acid the medium the more rapid is the destruction of hepatic cells."[6]

An allied process is often associated with fatty degeneration of the liver, which may be due to a variety of causes. The degeneration leads to physiological incompetence of the liver and loss of its antitoxic activities. Consequently the toxins continue in circulation and perpetuate the degenerative processes. It is not unlikely that the same sequence is the cause of death in delayed chloroform poisoning, as pointed out by Corner, who says:—

> "Between the administration of chloroform and the occurrence of delayed chloroform poisoning there is a vicious circle."[7]

At any rate, chloroform can undoubtedly produce similar fatty changes in the liver.

Tropical Liver.—It is well known that after each meal the liver undergoes a physiological enlargement due to hyperæmia, which in due course rapidly subsides. When, however, the *régime* of life disposes to excessive congestion or to actual attacks of recurrent gastro-hepatic disorder, the physiological post-prandial hyperæmia subsides less completely, and in course of time a chronic hypertrophy of the liver results, with a disposition to renewed congestion on slight provocation. Such is probably the explanation of the well-known "tropical liver," a disorder due to accidental chills after exposure to a high temperature in the tropics, especially in persons addicted to high living. The recurrent attacks in course of time lead to permanent and irreparable organic changes, and convert the liver into a *locus minoris resistentiæ,* which favours recurrence and an increase of the lesion.

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* Dr. Legge has pointed out a curious vicious circle associated with factories in which di-nitro-benzine is manufactured. The prevalence of toxic jaundice gave those factories so bad a reputation that only casual labour could be obtained, and such casual labour proved least resistant to the poison. Hence resulted increased illness and a further loss of reputation.—*Proc. Royal Soc. of Med.* (1917), X. (i.), p. 3.

Abscess of Liver.—Abscess of the liver results from infection by various micro-organisms, including the ordinary staphylococci and streptococci, *entamœba histolytica, bacillus coli communis, bacillus pyogenes fœtidus,* etc. Some of these organisms frequently reach the liver, but are destroyed by phagocytes, or washed away by the scouring biliary current. It is only in case of some hepatic insufficiency that they are able to multiply, such lowered power of resistance being due to dysenteric ulceration of the intestine, to alcoholism, to a tropical climate or other noxious influence. When once the micro-organisms, however, especially the *entamœba histolytica,* have succeeded in establishing a footing, they set up irritation, with the well-known phenomena associated with inflammation and suppuration, establishing what Cohnheim has called "a most pernicious *circulus vitiosus.*" The course of events in cases of inflammation and suppuration are so familiar that the briefest summary must suffice. The resulting phenomena are beneficent in purpose, and frequently succeed in triumphing over the invader. When, however, the resistance of the hepatic tissues is greatly reduced, or when the infection is abundant and the associated toxins are virulent, blood stasis takes place, the vessel-walls cease to be nourished, the contained blood coagulates, and the surrounding tissues perish. The products of disintegration then nourish the micro-organisms, which rapidly multiply and give rise to injurious toxins, thus further lowering hepatic resistance. The associated toxic products in their turn intensify the inflammatory processes, which spread by the breaking down and necrosis of liver cells. Copious exudation of serum is poured out, and in such serum float both the dead leucocytes that have perished in the struggle with the invaders, and the fresh leucocytes which are continually hastening to the field of battle. The resulting fluid known as pus tends to collect in an abscess cavity. Such pus, on the one hand, contains phagocytic cells and proteolytic enzymes, which assist the damaged tissue in its struggle for repair. On the other hand there are also present bacterial products which aggravate the inflammation. Moreover, the proteolytic enzymes, while useful in destroying necrosed tissues, are at the same time hurtful to adjacent hepatic tissues of low vitality, and cause their liquefaction. Hence it is that pus often causes rapid destruction of the damaged tissues, which in turn supply nutriment to the micro-organisms, and thus stimulate the formation of pus. In other words, pus feeds itself by liquefying the tissues round it, by perpetuating the very conditions in which it originated. Thus may be explained the enormous dimensions sometimes reached by abscesses of the liver.

Portal Cirrhosis.—Portal cirrhosis is another hepatic disorder that is complicated by a *circulus vitiosus*, although there is still difference of opinion as to the precise sequence of events. Formerly pathologists believed that the primary lesion in cirrhosis consisted in an overgrowth of the connecting tissues in the portal area, and that the degenerative changes in the liver cells were a secondary occurrence. According to this theory toxins act primarily on the portal sheaths, which become converted into dense bands of fibrous tissue, which crush or choke the hepatic cells out of existence. This destruction of the hepatic parenchyma then reinforces the primary toxæmia. In the words of Adami, "It is not impossible that a vicious circle is produced, and that irritants in the blood act through the vessels upon the fibrous structures of the portal sheaths, stimulating it directly to hyperplasia."[8] The modern view, however, is that the toxic substances act directly on the liver cells, the lesion being attributed to the sensitiveness or fragility of those cells when exposed to such noxious influences. Every hepatic cell which is undergoing degeneration owing to toxæmia may, in its turn, become a source of further intoxication. In other words, the products of cellular degeneration aggravate the primary disorder, and the hepatic toxæmia becomes a self-perpetuating condition. Fiessinger writes:—

> "Every increase of hepatic degeneration is followed by a partial absorption of the degenerated tissues. Such absorption naturally has a toxic action against which Nature protects itself by producing an anti-body or lysin which is antagonistic to the hepatic proteins. The process of cellular degeneration is consequently intensified since the parenchyma is exposed to a fresh poison. Thus a progressive circle is established. The more rapidly the morbid process advances the more virulent are the toxic influences that act on the liver."[9]

Rolleston thus sums up the process:—

> "As a result of the destruction of the hepatic cells and absorption of their proteins into the circulation hepatic anti-bodies or cytolysins are produced. These destroy the liver cells and thus a vicious circle is produced."[10]

Probably the safest conclusion is to steer a middle course, and to consider the irritating poisons which produce cirrhosis as affecting both fibrous and cellular elements in different ways.[11] In that case the resulting changes in one tissue may initiate fresh changes in the other tissue, or modify those already existing. Irritative hyperplasia in the one case and degeneration in the other may react on one another. A similar process occurs in renal cirrhosis.[12]

Cirrhosis also tends to be self-perpetuating, owing to several concurrent functional disorders. For example, a common complication is gastritis, the gastric catarrh manufacturing the toxins which are carried to the liver and provoke cirrhosis. This cirrhosis in its turn keeps up the gastric and intestinal catarrh.[13]

Cirrhosis again, owing to the associated portal engorgement, impairs the muscular and nervous activity of the bowel, and induces constipation. Such constipation in its turn leads to copræmia which tends to aggravate the cirrhosis.[14]

Cholelithiasis.—Some important circles are associated with cholelithiasis which is readily produced when catarrhal and infective processes lead to biliary stasis, so that the usual cleansing action of the biliary flow is lost. Gall-stones may form in any part of the biliary tract. For example, catarrh of the mucous membrane, especially of the finer bile ducts, may cause obstruction of bile, with increased desquamation of cells and an excessive formation of mucus. The morbid products are then apt to cause a precipitation of salts which form calcareous nuclei; these in their turn perpetuate the catarrh and aggravate the obstruction. Quincke writes:—

> "Icterus, in case it was not already present, results from the swelling of the mucosa of the bile-passages, causing obstruction to the outflow of the bile. In view of the narrowness of the bile-ducts, such an obstruction is readily produced either by swelling of the mucosa or by an increased desquamation of epithelial cells and an increased secretion of mucus, especially in the case of the finer passages. It is important to remember that the morbid products of a diseased mucous membrane may cause the precipitation of constituents from the bile which gradually form the nuclei of concretions. These, in their turn, keep up the catarrh and constitute an obstruction to the flow of bile, thus establishing a vicious

circle."[15]

Striking illustrations of this process occur during the growth of gall-stones in the gall-bladder. Here the ætiological factors include the stagnation of bile, bacterial infection and catarrh of the biliary passages, leading to a deposit of cholesterol, which forms a nucleus round which collect epithelial cells and bilirubin calcium. Such a nucleus may then act somewhat like a foreign body, producing irritation and desquamation of the mucosa, the products of which adhere to, and increase the size of, the nucleus. From such increased size result ingravescent irritation and catarrh, and so the process is accelerated. MacCallum thus describes the sequence:

> "All gall-stones contain a great deal of organic material derived from desquamated epithelial cells and coagulated albuminous matter as well as pigment. Many of them contain bacteria, and are formed in infected bile and within a gall-bladder which is inflamed, because, in this vicious circle, the presence of the stones aids in giving a foothold to bacteria, while they, in turn, through the inflammation they set up, aid in the growth of the stone."[16]

Wells also writes:—

> "When the bile stagnates in the gall-bladder, the cholesterol that is being constantly formed by the normal disintegration of surface epithelium accumulates until, even without infection, there forms a sediment of soft yellowish and brownish masses, consisting chiefly of cholesterol and bilirubin calcium. From this material calculi may eventually form, and, by their irritation, lead to further formation of cholesterol and increased growth."[17]

The reciprocal sequence of cholecystitis and gall-stones is especially common in typhoid fever, owing to the persistent presence of bacilli. This correlation may last for years after an attack of typhoid, and is doubtless a reason for the prolonged period during which persons convalescent after typhoid continue to be "carriers." Rolleston writes:—

> "Typhoid carriers not uncommonly have gall-stones, which no doubt favour persistence of the bacilli in the gall-bladder. A vicious circle thus results."[18]

In some cases of cholelithiasis the calculus blocks the outlet of the gall-bladder like a ball-valve. Hence results an accumulation of bile, and the greater the accumulation the more tightly is the outlet plugged and *vice versâ.*

Cholecystitis may also be a self-perpetuating disorder when the inflammation of the mucosa extends to the cystic duct and narrows its lumen. Such narrowing is apt to lead to retention and putrefaction of bile, and to increased narrowing, and so the process continues, until obstruction is complete.

Another circle is established in which cholecystitis and the resulting cholelithiasis, on the one hand, and gastro-intestinal catarrh, on the other hand, form the constituent factors. Thus Pepper writes:—

> "Dietetic and other indiscretions that lead to gastro-intestinal catarrh are of at least indirect influence in causing gall-stones, and a vicious circle is formed in that cholelithiasis and the antecedent cholecystitis . . . lead directly to gastro-intestinal catarrh, and, in virtue of the commonly associated precholecystic adhesions, induce much more serious disturbances of the stomach and intestines."[19]

Hepatoptosis.—Some interesting reciprocally acting correlations occur in hepatoptosis. In a normal abdomen the liver rests on a sloping shelf formed mainly by the lesser curvature of the stomach, the gastro-hepatic omentum, the pylorus and first part of the duodenum, part of the transverse colon, the right kidney and supra-renal body. This shelf, as Keith points out, is supported and pressed upwards by the tonus and contraction of the abdominal muscles, which maintain the liver in position. When, however, the abdominal muscles are lax or weakened, the shelf referred to above is no longer able to give adequate support, so that the liver tends to fall downwards and backwards. Such a displacement then further stretches the muscles and so the process is aggravated. Hepatoptosis, when once produced, may be worsened through kinking of the bile-duct and obstruction to the circulation through the hepatic veins and vena cava. Both such conditions add to the weight of the liver and so promote its increased descent.

Displacement of the liver is often secondary to tight-lacing and the wearing of corsets, and in turn perpetuates these practices. The pressure of stays, combined with the traction exerted by a heavy skirt at the waist tends to displace the viscera downwards. By this means the abdominal muscles are stretched and the suspensory ligaments of the liver are apt to become elongated; these conditions favour further displacement. Moreover the weakened abdominal parietes create a sense of laxity for which tighter corsets and lacing are resorted to.[20]

II.—THE NERVOUS SYSTEM.

Hypochondriasis.—The liver is closely connected with the nervous system both in health and disease. It is a familiar experience that mental worry or overwork disturbs the hepatic functions, which disturbance only too readily provokes further mental disorder or hypochondriasis. In other cases insufficient physical exercise is the primary factor, giving rise to hepatic torpor, the mental condition being secondary. In either case cause and effect aid and abet each other, the circulation of toxins being an important complication. Many cases of invalidism are due to this chronic disorder, and nothing but a holiday and complete cessation from business worries gives relief.

Neurasthenia. — Hepatoptosis has already been alluded to under the digestive system. But at times the hepatic displacement is closely connected with a condition of neurasthenia which may prove an obstinate disorder. The trouble is probably due to the fact that when the natural supports of the liver are withdrawn, the ligaments are called upon for support, and acute distress may be produced, accompanied by a sensation of dragging and discomfort. These symptoms are apt to arouse phobias, which provoke neurasthenia with lowered nervous and muscular tone, the sequence being especially common where there is a neuropathic predisposition. Mott thus describes the process:—

> "Any organ or structure which naturally or by habit performs its functions automatically, becomes disordered in its functions when there is mental apprehension and preoccupation regarding the performance of that function. This is an everyday experience. So we can understand how a continued preoccupation regarding the function of an organ like the heart or the stomach, or, indeed, any organ of the body, of the perfect action of which we should be quite unconscious, becomes an obsession; for disordered function tends to repercussion in the field of consciousness, causing a continual conflict between reason and emotion for readjustment. Thus a vicious circle is liable to be established, owing to the inherent state of emotivity of the neurasthenic." [31]

In other cases, the muscular weakness is the most prominent symptom, the sequence being hepatoptosis, neurasthenia, loss of muscular tone, hepatoptosis.

Rickets.—Rickets is another disorder in which, according to some authorities, the nervous system and the liver are concerned. Pritchard thus describes the correlations:—

> "The pathogenesis of the disease thus defined must be regarded as a vicious circle of events in which each individual link in the chain acts and reacts on the others. In describing this vicious circle it is difficult to know at what point to begin; indeed, in the production of rickets there is no starting-point; the disease may start in a variety of ways.
>
> "The vicious circle may be briefly described as consisting of an inefficient liver associated with a disturbed central nervous system acting and reacting on one another.
>
> "These are the two pathological pivots on which, according to my view, the symptoms mainly hinge.
>
> "The liver may be primarily damaged by faulty methods of feeding, from abuse of its functions, or from overwork, or it may be inherently or congenitally weak. As a rule, there is first indigestion, then efforts on the part of the liver to oxidize or destroy these products, then collapse or failure of liver function with the escape of these unoxidized products into the general circulation, and finally poisoning of the nervous system itself.
>
> "Poisoning of the nervous system, from whatever cause induced,

not only intensifies the liver inefficiency by interfering with its nervous mechanism, but it also leads to incapacity of other furnaces in the body, and herein lies the essence of the vicious circle of events."[22]

III.—THE VASCULAR SYSTEM.

The heart is dependent for its healthy activity on the purity of the blood transmitted by the liver. If the liver fails in its duties as a catchpit, that is, allows toxins that should be destroyed to reach the heart, the myocardiac efficiency will be impaired; both its suction and its propulsive force will suffer. Hence the flow of venous blood will be retarded, and this in turn will be followed by hepatic engorgement, thus further interfering with antitoxic activities. This cardio-hepatic circle is even graver when the disorder starts from obstructive, especially mitral, disease. The hepatic symptoms are at times merely added to the cardiac. At other times the hepatic symptoms are dominant, producing a condition to which the term "hepatic asystole" has been applied. Barié writes:—

> "In cases of hepatic asystole the hepatic functions are disordered, and the disorder aggravates the cardiac insufficiency. In other words, the hepatism associated with cardiac failure creates a vicious circle as a result of which the heart and liver react on one another."[23]

These pernicious correlations may be observed in many cases of cardiac failure. There may be a persistent "morbid equation" which at length terminates fatally.

IV.—THE RENAL SYSTEM.

The liver and kidneys are the principle emunctories of the body, and are closely associated by a physiological synergy. In the healthy organism there is an ever-active *circulus virtuosus*, owing to which products of metabolism are so transformed in the liver cells that they can readily be excreted by the kidneys.

The renal activity in its turn so purifies the blood that is returning to the liver that this organ can satisfactorily carry out its metabolic duties. Hepatic and renal functional activities are mutually dependent on each other.

In disease there is a corresponding pathological solidarity; in other words, a *circulus vitiosus* is established, at any rate when the disorder is so severe that the vicarious relief which one organ can give to another is overtaxed. Hepatic insufficiency then breeds renal insufficiency, and *vice versâ.*

These injurious correlations are present in some of the forms of hepatism referred to above; in fact, it is often difficult to disentangle hepatic from renal conditions.

Eclampsia affords a good example of a disease in which sometimes the liver, sometimes the kidneys, is primarily at fault. If the liver is mainly affected, profound changes take place in the hepatic parenchyma, characterized by areas of necrosis, hæmorrhages, and fatty degeneration of the liver substance. The loss of the hepatic antitoxic functions allows the toxins to continue in the circulation, in which case the functional activity of the kidneys is depreciated, and so the process continues. Wells writes:—

> "A reasonable view of the cause of eclampsia is that it is initiated by the excessive products of metabolism thrown into the blood of the mother, both from the fœtus and from her own over-active tissues. These cause injury to the kidneys, leading to a further retention, or injure the liver so that the normal metabolic processes of that organ (particularly oxidation) cannot be carried on; or, perhaps more often, both liver and kidney as well as other organs are injured. In this way a vicious circle might be established and rapidly lead to an overwhelming of the maternal system with toxic products derived from both her own and the fœtal tissues."[24]

The liver may be unable to destroy toxins, while the kidney may be unable to eliminate them. Consequently the primary toxæmia is aggravated by a retention toxæmia.

THE BREAKING OF THE CIRCLE.

Disorders which are complicated by a *circulus vitiosus* often form a *crux medici*. Reactions perpetuate, in lieu of relieving, the primary evil, and the morbid process thus tends to become self-aggravating, and frequently ends fatally.

Although, however, the disorders so complicated tend to chronicity and self-aggravation, unaided Nature sometimes succeeds in effecting a cure, or at any rate in arresting the progress of the disease.

Thus, attacks of so-called "biliousness" may be self-curative through a temporary anorexia, which allows time for the readjustment of disturbed gastro-hepatic correlations. In severe cases, a violent emesis or diarrhœa may remove the irritating matters from the stomach, while the associated pressure on the liver helps to relieve the biliary passages of their stagnating secretions. At other times, a sub-conscious desire for increased exercise acts in the same direction.

Even so chronic a disorder as gall-stones may be successfully overcome. For many a long year cholecystitis and cholelithiasis may have been maintaining each other. All at once peristalsis of the walls of the gall-bladder may set in, and expel the calculus that has been causing the disorder. Even so self-perpetuating a condition as an hepatic abscess is occasionally arrested by absorption of the pus.

In cases of cirrhosis Nature often succeeds in arresting the morbid process by an extensive hyperplasia of healthy hepatic tissue, and by the development of a collateral circulation. Both these methods are as a rule only makeshifts, but they indicate an attempt at repair which succeeds in postponing a fatal issue.

But Nature is not always so successful. Her methods are frequently ill-adapted to the end in view, involve pain and loss of time, and may fail even after a long attempt. The *ars medica* is usually called for if rapid success is to be obtained; the detailed therapeutics would require an article to themselves. Whether by modifying the *régime* of life, by mechanical supports, by pills and potions, or by the knife, the *ἰατρός πολύπιυρος* "the experienced physician" often succeeds in breaking the circle.

"Art triumphs where Nature fails."

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A Fatal Case of Dysentery in an Adult caused by Morgan's Bacillus.

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MRS. E., æt. 36, first came under my care on December 6, 1919. She complained of an attack of "piles," accompanied by diarrhœa and abdominal pain. Parturition was expected at any moment, and not much notice was taken of her symptoms, especially as there was no rise of temperature or pulse rate, and her appearance was good. Next day, December 7, 1919, the patient was delivered of a normal male child without assistance or any complication. Diarrhœa persisted, indeed became more frequent. The stools were a deep yellow, very offensive, and of the consistency of thick cream. It was then ascertained that diarrhœa had preceded the birth of the child for twelve days, with three or four stools per diem. These now became more frequent, about ten a day, of light ochre colour, fluid, very offensive, and of the pea soup variety. Widal's and Wassermann's reaction were negative. The temperature varied between 99° and 100°. There was a good deal of distension, and pain (not very acute) was complained of above the umbilicus extending down the left flank. Catechu, opium, and bismuth were given by the mouth, and strong starch and opium enemata by the bowel. These only gave temporary relief. Fluids and semi fluids were taken by mouth only. No vomiting.

December 13, 1919, six days after parturition the motions were practically continuous day and night, and were all of the same consistency and colour. The temperature never rose above 100°, the pulse varied from 100 to 112. The abdomen was now more distended and tympanitic. On palpation, nothing could be felt. Pain was elicited by deep pressure at the level of the umbilicus and extending down the left flank. No point of pain on pressure over the appendix, nor rigidity of muscles. The tongue was dry and covered with brown sordes, and the patient had an almost typical typhoid facies. The enemata were continued and alternated with silver nitrate (1 in 1,000) enemata. A specimen of fæces, at this stage, was examined by one of the best London pathological institutes, the report reaching me two days after the patient's death. It stated that there was no evidence of typhoid fever or paratyphoid. Influenza bacilli, dysenteric amæbæ or dysenteric bacilli were not detected; but organisms giving the cultural and staining reactions of Morgan's No. 1 or No. 14 Bacillus were present. On December 20, in spite of all stimulation, transfusions, etc., the patient sank, and died early on December 22, 1919.

POST-MORTEM.

Every abdominal organ was found normal, including the appendix and cæcum, with the exception of the transverse colon. This at first sight was thought to be a large abscess cavity, extending across the abdomen, at the level of the lower costal margin, adherent to and covered by omentum, but not adherent to the parietes. The portion of bowel involved extended from the hepatic flexure to the splenic flexure; before and beyond these parts the bowel appeared normal, internally and externally. Upon opening the transverse colon, a large amount of dark grey opalescent fluid escaped like dirty pus. The interior was much disorganized and unrecognizable, friable with a multitude of large and small ulcers eating through the muscular coats of the intestine and adherent to the omentum. No actual perforations had taken place. Apart from this involved portion of the colon no peritonitis was present.

Practical Notes.

The Functional High Temperatures of Childhood.

H. Jumon points out that there is very little attention given in the text-books to fever as an isolated symptom, although in children nothing is more common than a temporary rise of temperature or a prolonged sub-febrile condition, the ætiology of which is not at all apparent. Many names were given in former days to such conditions, such as herpetic fever, the fever of dentition or of growth, lymphatic fever, adenoid fever, and so forth, evidence of the trouble taken by the clinician to link up the symptom to a definite cause.

The cause of a feverish or sub-febrile condition is usually attributed to a latent tuberculosis, so that from the point of view of diagnosis, and especially of prognosis, it is of real importance to try and prove that such is not the case, but that the fever may be caused by adenoids, glands, gastric troubles, and the like, whilst in some cases it may be purely physiological. To the latter the term functional, essential, or physiological pyrexia is applied. Jumon prefers the term hyperthermia, because, as Renault points out, there can be hyperthermia without fever, that is, a high temperature without general malaise, loss of appetite, and gastric troubles. These rises of temperature can only be understood properly when the thermic reactions of the healthy child are known. These reactions are as follows, the data being the rectal temperatures, the only exact method in the child :—

1. *The nursling.*—The new-born infant has a temperature of 37°·7, which remains constant throughout the 24 hours. The variations are at most two- or three-tenths at night.

2. *From five months of age until the child walks.*—There is a morning minimum and an evening maximum, after one year old, 36°·7 in the morning and 37°·4 in the evening. Krantz gives rather a bigger range, 36°·4 to 37°·1 in the morning and 37°·5 to 38° in the evening.

In the adult the range is from 36°·6 to 36°·8 in the morning, 37°·2 to 37°·4 in the evening. The normal adult has attained equilibrium, whereas the child is only working towards that stage; and in view of the hyperexcitability of the child's nerve centres, and the intensity of its organic reactions in response to all sorts of external stimuli, it is not surprising that even a prolonged rise of temperature may, in certain cases, be set down as physiological.

I. *Hyperthermia due to movements.*—The temperature of the normal child is raised by activity. A walk of from four to five kilometres raises the temperature to 37°·8, which lasts for about three-quarters of an hour to an hour. A diagnosis of latent tuberculosis can only be entertained if the reaction exceeds the physiological limits, or if the rise of temperature continues for more than one hour in spite of rest. The normal reaction is one of fatigue, to which no importance can be attached.

II. *Hyperthermia arising from digestive activity.*—The temperature of the nursling is not affected by meals except, in some cases, when the mother has a menstrual period. After the age of one year, the temperature falls a quarter of an hour after a meal, and then rises from four- to six-tenths. The nature of the food taken may have an effect.

III. *Hyperthermia of various origins.*—The children of arthritic parents may have what is termed arthritic fever without assignable cause. Lomby thinks this may be the equivalent of an attack of gout. Febricula may be caused by thyroid instability, and is remedied by opotherapy. Nervous children are more subject than others to sudden rises of temperature.

A diagnosis of functional hyperthermia should only be made after scrupulous examination of the child has excluded all pathological rises of temperature of obscure origin, such as the early stage of an eruptive fever, pneumonia with the physical signs late or absent, malignant endocarditis with a long prodromal pyrexial stage, mastoiditis, erythema nodosum, infantile paralysis, or osteomyelitis. In the absence of these, when fever is the only symptom, the diagnosis lies between a latent tuberculosis, an adenoid or glandular infection, digestive troubles, and convalescence from an acute febrile disease.

I. *Latent tuberculosis* may show itself (1) by the pre-tuberculous bacillary fever which has an acute and prolonged course, showing the typhoid curve; (2) by a simple febrile type in which the temperature oscillates between 37°·5 and 38°·5. This may be present for from two to four weeks before any physical signs can be heard in the chest, a false defervescence without crisis occurs; (3) by irregularity in the temperature during repose after fatigue, or by persistence for more than an hour after being raised by walking.

II. *Adenoid or lymphoid hyperthermia.*—Many attacks of febricula originate from the naso-pharynx. A persistent sub-febrile condition, lasting for months, or for years, may be caused by enlarged tonsils, adenoid growths, coryza, or rhino-pharyngitis. Even for some time after removal of these causes, there may be an evening rise to 38°.

III. *Sub-febrile conditions of digestive origin.*—The temperature rises after meals. and the child has anorexia, great thirst, fœtid stools, and general dyspeptic conditions. Chronic entero-colitis with habitual constipation has the same symptoms. Arthritic children, who have had digestive troubles in early infancy, are often attacked with pruriginous dermatoses, headache, and insomnia. The fever is due to intestinal auto-intoxication, and every sort of food may temporarily be the active cause. The commonest type of fever is intermittent, the increase in weight is irregular, and the urine contains indol, phenol, and scatol.

IV. In susceptible children, the temperature may often remain irregular after convalescence from infectious diseases.—(*Le Bulletin Médical*, April 17, 1920.)

Reviews of Books.

Diseases of the Ear in School Children, an Essay on the Prevention of Deafness. By James Kerr Love, M.D., F.R.F.P.S.G Pp. viii + 94. Bristol : Wright and Sons, Ltd. 5s. 6d. net.

The final sentence of this excellent little volume thus proclaims its aim : "I have tried to show that the 'Passing of Deafness' is not a Utopian, but an eminently practical thing." A perusal of the whole volume (and once it is opened, the reader finds it difficult to relinquish until it has been read from cover to cover) leaves a conviction that he has fully proved his contention. A record is furnished of three years' work done for the Glasgow School Board in 1912–15.

Suppurative and non-suppurative ears are dealt with in the first four chapters, and it is insisted upon that, beyond tubercle and deaf-mutism, the amount of ear disease in the pre-school age is small; it is in school age that the ears are attacked. Hence efficient treatment during that period is a paramount necessity, and the author proves this up to the hilt. The treatment must, however, be efficient. *Nurses must be trained and the supervision must be by a specialist.*

Chapter IV. deals specially with syphilitic deafness, Kerr Love's work in which has been epoch-making. In regard to hearing tests, the eminently practical one of the whisper at six feet is advocated. Chapter VI. is occupied by a discussion upon hereditary deafness and its relations to the Mendelian theory, and the author then passes to educational arrangements and general considerations. It is to be noted that the author considers that hereditary deafness treated upon Mendelian principles for two or three generations would become a curiosity.

The book is one for every otologist and every school doctor to read, and London school authorities should follow in the steps of their Glasgow colleagues.

X-Ray Observations for Foreign Bodies and the Localization. By Captain Harold C. Gage, A.R.C., O.I.P. London : William Heinemann.

This little work was written, so the author informs us, in June, 1917; and it is a pity, from his point of view, that it did not appear in that year, for its circulation would doubtless have been greater than will now be the case. It is, however, very important that knowledge gained in the war should be made readily available for post-war civil practice, and the existence of a comprehensive monograph on localization in book form will be of permanent value. It is scarcely likely to get out of date in peace time, for nearly four and a half years of war failed to bring forth any *principle* which had not previously been made use of.

So far as the reviewer is able to judge, practically all methods of any value are adequately described, though they are often credited to people who were by no means their originators, or even the first publicly to advocate them.

(We would have liked to see credit given to Lyster for the use of malleable metal strips to outline the contour of the body; and to Shenton for the parallax method.)

The author does not seem to have grasped the extreme importance of all instruments used in contact with the patient being readily sterilizable. For example, he describes Hernaman-Johnson's "Rery-Localizer" in its original improvised form—"a metal ring on the end of a piece of wood"—whereas this instrument was placed on the market with an aluminium handle, so that it could be readily boiled. Neither is any description to be found of a sterilizable aluminium case for use with the Read flarescope or crystoscope.

There is an excellent chapter on the localization of foreign bodies in the eye, and the telephone probe and the Berganié vibrator are described.

On the whole, this little volume may be confidently recommended as a work of reference which should be on every radiologist's shelves and which most surgeons should peruse.

A Text-book of Physiology. By MARTIN FLACK, C.B.E., M.B., CH.B., and LEONARD HILL, M.B., F.R.S. Pp. viii + 800. London: Edward Arnold. 25s. net.

THIS text-book of physiology, though written primarily to meet the requirements of the medical student, contains much matter concerning the practical applications of physiology to general medicine, and should prove of value to the general practitioner. It begins with a consideration of general biological principles and of the general biology of the cell, which is followed by a physico-chemical introduction; the latter should prove very useful. A general account of the chemical composition of the body and of enzyme action follows, after which the general physiology of the body is considered. The descriptions throughout are clear and as brief as circumstances permit, and the text is copiously illustrated with drawings and diagrams of considerable merit. With regard to the various sections, those dealing with respiration, body temperature, and general metabolism and dietetics are particularly well done. Under the nervous system, the functions of the various structures are clearly and succinctly described; the autonomic nervous system being dealt with in a separate chapter. Under the ductless glands, no reference is made to the function of the parathyroids in the regulation of guanidine metabolism and production of tetany. It is stated that the frog ovum "fertilized" by pricking does not develop beyond the tadpole stage; this is incorrect. There is some confusion in the description of the precipitin test for blood (p. 113). The polymorphonuclear leucocytes are certainly not 18 microns in diameter (p. 97). Altogether this new physiology is an admirable one, readable, well produced and a valuable addition to medical text-books.

Typhoid Fever, considered as a Problem of Scientific Medicine. By FREDERICK P. GAY, Professor of Pathology in the University of California. Pp. xiv + 286. London and New York: The Macmillan Company.

IN this treatise the author's aim has been to obtain an insight into the nature of the problem of typhoid fever as a whole, by following the life history of the bacillus rather than the manifestations of the disease it produces. It is, beyond question, one of the most valuable expositions on the subject that have appeared. It deals most thoroughly with every aspect *ab ovo usque ad mala,* and presents a well-balanced judgement throughout. Every investigator into the subject will not only find the book a necessity, but will have reason to be infinitely grateful for the full bibliography which puts the crowning touch to the work. The value of this is indicated by the fact that it takes up no less than forty pages.

Kala-azar, its Diagnosis and Treatment. By ERNEST ROGERS, C.I.E., I.M.S. Pp. viii + 39. Calcutta and London: Butterworth & Co. Rs. 2 net.

THIS little book gives a good account of kala-azar, its diagnosis and treatment, and the reliability of the contents is vouched for in a foreword contributed by Sir Leonard Rogers.

In the earlier pages the salient features of the disease are described. Clear and full directions are given for the performance of splenic puncture, so important for diagnosis, and by adopting the procedure detailed all risk is practically abolished. A table of the differential diagnosis is included.

As regards treatment, full directions are given for the intravenous administration of antimony, which is practically a specific. The author prefers sodium antimonyl tartrate to ordinary tartar emetic (which is the potassium salt). The treatment of complications is also dealt with.

As an adjunct to antimony the author speaks highly of injections of a mixture of turpentine, creasote, camphor, and olive oil in certain circumstances; for example, in acute cases and in cases in which the spleen remains large and hard, as a preliminary to antimony.

The book is illustrated with four plates showing patients, methods of splenic puncture, and intravenous injection.

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